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YELLOW FEVER.

JOHNSON (W. B.). **Recent Advances in the Knowledge of Yellow Fever.**—*Brit. Med. Jl.* 1932. Aug. 13. pp. 285–288. [14 refs.] Discussion pp. 324–325.

A general summary of the pathology and epidemiology of the disease with special reference to the work of the Rockefeller Commission in West Africa. Of special interest are notes showing that the dry northern regions of Nigeria are both infectible and also able to maintain infection throughout the year. The methods of attacking the disease in West Africa are based upon the following lines :—Institution of pipe-borne water ; education in hygiene by means of lectures and newspaper articles ; training African sanitary and medical personnel and their dissemination throughout the country.

The discussion centred largely around the application of recent studies on yellow fever to measures for its control, including the agreement for controlling the spread of infectious disease by aircraft. Col. F. P. MACKIE, I.M.S., mentioned mouse protection experiments with 53 sera collected by Col. KING in Madras ; out of these five gave some degree of protection, including two definitely positive sera.

E. Hindle.

LEAGUE OF NATIONS MONTHLY EPIDEMIOLOGICAL REPORT. 1932. Mar.–Apr. Vol. 11. No. 3–4. pp. 79–82. **Yellow Fever since the Beginning of 1931.** [In parallel French & English.]

The reappearance of yellow fever in West Africa during 1931 in a number of scattered localities with no connexion between them (see Fig.) can only be explained on the supposition of latent yellow fever foci in these countries during the intervals between epidemics, and the experiments carried out in Nigeria and Sierra Leone by means of protection tests [see this *Bulletin*, Vol. 28, p. 283] support this view. In Sierra Leone, which used to be considered one of the worst foci of yellow fever, no cases have been reported since 1910; yet in protection tests in 1929 on 34 children between 4 and 10 years of age, one gave complete and three partial protection, showing that the disease was still present.

Localities of Western Africa in which Yellow Fever cases were reported in 1931 and during the first four months of 1932. [According to BOYÉ (below) Yellow Fever cases have also occurred in 1931 at Korango and Kayo (French Soudan) and at Dosso (French Niger Territory).]

[Reproduced from the *League of Nations Monthly Epidemiological Report.*]

The recrudescence of the disease in Brazil, particularly in Ceara, and on the coast of Colombia, near Cierraga, is also evidence of its persistent nature. The number of cases in both Africa and America is very small, but epidemiological observations indicate that the cases notified have no numerical value, but only an index value. The widely scattered localities from which the cases have been recorded are a clear indication of the potential danger of this disease. *E. H.*

BOYÉ. Les cas déclarés de fièvre jaune chez des Européens en Afrique Occidentale Française en 1931. [**Notified Cases of Y. F. among Europeans in French West Africa during 1931.**]—*Bull. Office Internat. d'Hyg. Publique.* 1932. June. Vol. 24. No. 6. pp. 943-947. With 1 map.

During 1931, cases of yellow fever have occurred in Togoland and in six out of the nine French colonies in West Africa, viz.: Ivory Coast, Upper Volta, Soudan, Senegal, Niger Territory and Mauritania. A total of 40 cases in Europeans was observed of whom 33 died. The cases occurred from June to December, the highest number being in July (16). It is proposed to establish a special laboratory in French West Africa for the scientific diagnosis of suspicious cases, and also to continue the delimitation of endemic areas. *E. H.*

HUGHES (T. P.) & SAWYER (W. A.). **Significance of Immunity Tests in Epidemiology as illustrated in Yellow Fever.**—*Jl. Amer. Med. Assoc.* 1932. Sept. 17. Vol. 99. No. 12. pp. 978–981. [11 refs.]

Using the intraperitoneal protection test in mice the authors have examined the sera of a number of persons from countries where yellow fever has never existed in order to test the specificity and reliability of the test. The sera of 88 Chinese aged from 21 to 53 living in Peiping, who had never been out of China, all gave negative results with the exception

of a few inconclusive ones. Out of 109 Canadian residents, one serum gave protection because of some property in the serum itself and five gave inconclusive results. The remainder were all negative. On the other hand the sera of 21 persons of the same British stock, who had either recovered from yellow fever or been immunized by vaccination, all gave complete protection. The sera of 20 American negroes from near Annapolis gave no protection in 19 and inconclusive results in one. Ninety-six sera from Panama and the Canal Zone gave complete protection in 14 cases, 5 inconclusive and 77 no protection. All the sera giving protection came from persons of 30 years of age or older. None of the 55 sera from persons under 30 years of age gave protection, which agrees with the history of yellow fever in the Isthmus, the last case having been recorded in 1905. It is evident that this test is highly specific and furnishes a very useful indicator of past infections.

E. H.

Durieux (C.). Mission d'enquête amarile au Niger et au Dahomey. [**Yellow Fever Mission to French Niger and Dahomey.**]—*Bull. Soc. Path. Exot.* 1932. July 6. Vol. 25. No. 7. pp. 834-836.

The examination of sera for protection against yellow fever at Zinder gave 44 per cent. positive (actual numbers not given) among the adults but all negative among children, the youngest positive case being 17 years of age. At Tessaoua there were 48 per cent. positive, the youngest aged 12 years. At Agadès the sera (86) were all negative, except that of a child who may have visited other districts. *E. H.*

Soper (Fred L.), Frobisher (M.), Jr., Kerr (J. A.) & Davis (N. C.). **Studies of the Distribution of Immunity to Yellow Fever in Brazil. I. Postepidemic Survey of Magé, Rio de Janeiro, by Complement-Fixation and Monkey-Protection Tests.**—*Jl. Preventive Med.* 1932. Sept. Vol. 6. No. 5. pp. 341–377. With 5 figs. [34 refs.]

Using an antigen prepared from the livers of monkeys infected with yellow fever, the authors made complement fixation tests on 300 sera collected in Magé and Santo Aleixo, and on 101 samples from Piracicaba, lying outside the yellow fever zone. Approximately 42 per cent of the sera from Magé county were positive as compared with only 3 per cent. of those from Piracicaba. A small number of tests made on the same individuals at intervals of four, sixteen and twenty-two months after an epidemic, seemed to indicate that there is a tendency, especially the first year after infection, for the titre of the complement-fixing bodies in the blood to decrease.

Comparative results of complement fixation and monkey protection tests with 76 sera showed that as a rule positive sera give complete protection, but about 13 per cent. showed a negative reaction and yet gave protection. It would seem that the complement-fixing bodies are less constantly produced and also less permanent than the protection bodies. It is of interest that a patient who had yellow fever in April 1929, and failed to react clinically to repeated feedings of infected mosquitoes over a period of eighteen months, gave the following results in monkey protection tests during 1929—May and July, prevented death but not fever; September, no protection; October, full protection; November, severe fever and recovery; December, full protection.

The results of the tests indicate that at the time of the outbreaks of yellow fever in Magé and Santo Aleixo a relatively large percentage of the inhabitants acquired immunity without any apparent attack of the disease, a factor which must be taken into account in any study of the distribution of the disease.

The influence of variations in metabolism and diet on the course of the infection is considered a possibility in view of the protective action of certain food substances against liver destruction caused by such poisons as carbon tetrachloride and chloroform. In this connexion some of the highest death rates have been obtained in closed groups which had been subjected to uniform diets probably well below optimum. *E. H.*

MONTEIRO (J. Lemos) & TRAVASSOS (J.). Diagnostico sorologico da febre amarella. Sobre a reacção de fixação do complemento. [**Serological Diagnosis of Y. F.: Complement Fixation.**]—*Mem. Inst. de Butantan.* 1930. Vol. 5. pp. 173–191. With 1 graph. English summary.

By means of complement fixation [see this *Bulletin*, Vol. 27, p. 878] using yellow fever liver antigen, a number of sera of Brazilians have been tested. The sera of natives living in endemic areas were found to contain complement fixing antibodies in about 50 per cent. of the cases, whilst those of newly arrived foreigners were negative. The sera giving complement fixation were also found to protect rhesus monkeys against the infection. *E. H.*

LEFROU (G.). Vieux documents sur l'examen des urines dans la fièvre jaune. [**Old Records of the Examination of Urine in Y. F.**]—*Bull. Soc. Path. Exot.* 1932. June 8. Vol. 25. No. 6. pp. 535-539.

The author calls attention to the work of earlier writers on this subject, especially DA COSTA ALVARENGA who wrote an account of the Lisbon epidemic in 1857, and called attention to the importance of albuminuria in the diagnosis and prognosis of yellow fever. DECOREIS also gave an excellent account of the changes in the urine based on a study of the epidemic at St. Pierre (Martinique) in 1880 and 1881.

E. H.

KERR (J. A.). **Studies on the Transmission of Experimental Yellow Fever by** *Culex thalassius* **and** *Mansonia uniformis*.—*Ann. Trop. Med. & Parasit.* 1932. July 14. Vol. 26. No. 2. pp. 119–127. [12 refs.]

Culex thalassius, which occurs throughout the coastal zone of tropical and South Africa, breeds in a great variety of places, including the shallow brackish or salt water at the edges of lagoons. The author finds that it is capable of transmitting yellow fever by its bite after an extrinsic incubation period of 12 to 18 days and remains infective for 27 to 49 days after the infecting meal. As the density of this species is known to be very great at certain places on the coast it may be of some significance in the epidemiology of yellow fever.

Mansonia (*Mansonioides*) *uniformis* seems to be unable to transmit the virus of yellow fever by its bite, after an extrinsic incubation

period of 21 days or less, but the virus is able to persist in the body of the insects for at least 24 days and possibly throughout their lives; consequently it cannot be entirely excluded as a possible factor in places where it is abundant. *E. H.*

KUMM (Henry W.) & FROBISHER (Martin), Jr. **Attempts to transmit Yellow Fever with Certain Brazilian Mosquitoes (Culicidae) and with Bedbugs** (*Cimex hemipterus*).—*Amer. Jl. Trop. Med.* 1932. Sept. Vol. 12. No. 5. pp. 349–361. [14 refs.]

Culex corniger, *C. coronator*, and *C. surinamis*, in captivity, would not bite either human beings or monkeys. *Joblotia digitata* was fed with difficulty on infected monkeys, and the only two specimens known to have engorged on blood containing yellow fever virus failed to infect or immunize normal monkeys injected with their contents after intervals of 14 and 20 days respectively. *Mansonia titillans* fed on infected monkeys was apparently incapable of transmitting yellow fever by bite, but the virus remained alive and monkeys inoculated with the contents of mosquitoes at various intervals up to a maximum of 30 days after the infecting meal, all became infected and died of the disease.

When bed-bugs, *Cimex hemipterus*, were fed on yellow fever monkeys, the virus died off so rapidly that the contents failed to produce infection later than the second day after the infecting meal. It is just possible that the faeces contained active virus during the first and second days, but not later; consequently the authors are unable to confirm the results of MONTEIRO [see this *Bulletin*, Vol. 27, p. 485]. *E. H.*

KERR (J. A.) & HAYNE (T. B.). **On the Transfer of Yellow Fever Virus from Female to Male** *Aëdes aegypti*.—*Amer. Jl. Trop. Med.* 1932. May. Vol. 12. No. 3. pp. 255–261.

A careful series of experiments testing the possibility of the transfer of yellow fever virus from infected females to normal males of *Aëdes aegypti*. Large numbers of mosquitoes were used and the males brought in contact with the females immediately, three weeks and nine weeks after they had taken infecting blood meals. Infectivity tests with 152 of these males gave no evidence indicating the presence of yellow fever virus and the results of immunity tests confirmed these results. It is evident, therefore, that the transfer of yellow fever virus from mosquito to mosquito without passage through a vertebrate host, is of no importance in the epidemiology of yellow fever [see also this *Bulletin*, Vol. 28, p. 285]. *E. H.*

DAVIS (Nelson C.). **The Effect of Various Temperatures in modifying the Extrinsic Incubation Period of the Yellow Fever Virus in** *Aëdes aegypti*.—*Amer. Jl. Hyg.* 1932. July. Vol. 16. No. 1. pp. 163–176. With 1 chart. [11 refs.]

An account of experiments confirming previous observations [see this *Bulletin*, Vol. 28, p. 285] on the influence of temperature on the development of yellow fever virus in *Aëdes aegypti*. The Asibi (West African) strain of virus and a South American race of mosquitoes were used throughout. Ability to infect was acquired after four days

at 37°C. ; after five days at 36°C. ; and after six days at 31°C. ; after eight days at 25·1°C. ; and at 23·4°C. they were infective after eleven, but not after nine days. At 21°C. they became infective after eighteen days. When kept at 18°C. they were not infective after thirty days, but after an additional six days at room temperature (23·4°C.) became infective, showing that there had been some development of the virus, or progression towards infectivity, even at the lower temperature.

There was no attenuation of the virus in mosquitoes at low temperatures for, even after storage at 8°C., they became infective when placed at 36°C. for six days. The author re-examined the protocols of FINLAY'S total of 103 experiments in which he claimed to have proved the transmission of yellow fever by the bites of mosquitoes. In only two experiments, where transmission was claimed, had the mosquitoes received their infective meal from patients as early as the third day of fever, and in both these instances the volunteers for the experiments were bitten after an incubation period in the mosquito of only two days. Even with the most favourable interpretation it is impossible to admit transmission to any of his experimental subjects, unless some of the wild mosquitoes he used had been previously infected in nature. *E. H.*

KUMM (Henry W.). **Yellow Fever Transmission Experiments with South American Bats.**—*Ann. Trop. Med. & Parasit.* 1932. July 14. Vol. 26. No. 2. pp. 207–213.

Since *Aëdes aegypti* readily feeds on bats the possibility of these animals serving as a reservoir of yellow fever virus has been investigated. Attempts to transmit the disease to *Molossus obscurus* Geoffroy, a common bat in houses in Bahia, and *Desmodus rotundus* Geoffroy, a common vampire bat of South America, by means of the bites of infected mosquitoes, and later to reobtain the virus, gave entirely negative results. In two experiments, when vampire bats were allowed to bite an infected monkey and after about one minute's interval bit a normal monkey the latter became infected with yellow fever. Such mechanical transmission has probably little importance in the epidemiology of the disease in human beings, but in the case of animal diseases may be of economic importance. *E. H.*

SELLARDS (A. W.) & LAIGRET (J.). Contrôle, par épreuve sur *Macacus rhesus*, du pouvoir protecteur du sérum des hommes vaccinés contre la fièvre jaune avec le virus de souris. [**Control Tests with *M. rhesus* on Protective Value of the Serum of Human Cases Vaccinated against Y. F. with Mouse Virus.**]—*C. R. Acad. Sci.* 1932. June 13. Vol. 194. No. 24. pp. 2175–2178.

The blood of three patients vaccinated with small doses of mouse virus [see this *Bulletin*, Vol. 29, p. 572], has been tested in rhesus monkeys and found to possess a high degree of immunity, not only against mouse virus, but also against the ordinary human virus maintained by passage in monkeys. *E. H.*

SAWYER (W. A.), KITCHEN (S. F.) & LLOYD (Wray). **Vaccination against Yellow Fever with Immune Serum and Virus fixed for Mice.**—*Jl. Experim. Med.* 1932. June 1. Vol. 55. No. 6. pp. 945–969. With 1 text fig. [24 refs.]

A valuable account of experiments on the method of vaccination against yellow fever by the use of immune serum and mouse virus.

Experiments with virus exposed to the action of formaldehyde or tricresol showed that mixtures exposed for only a short time to the attenuating agents produced yellow fever in the monkeys inoculated, and those exposed for a long time did not protect. Between these extremes vaccinated animals were found to be immunized without showing any fever, but the zone of exposure to the attenuating agent was found to be inconstant and narrow. It is well known that a simultaneous injection of virus and immune serum confers protection without producing any signs of infection, and this method was finally adopted as the most satisfactory, using mouse virus instead of the more virulent monkey strain. Virus mixed with immune serum was found to be slowly neutralized at room temperatures but this process was stopped by drying and a mixture kept dry in a sealed tube at room temperature for eight months retained its immunizing power. When yellow fever virus is completely neutralized by immune serum *in vitro* it loses its power to immunize or infect. The degree of immunity produced by the inoculation of mouse virus and immune serum was found to be almost as high as that following the naturally acquired disease. It was necessary, however, not to use an excessive quantity of immune serum as this greatly diminishes and possibly prevents the production of antibodies in the blood after injections of virus.

The method finally adopted was a single injection of a dried mixture of living yellow fever virus fixed for mice, and human immune serum, with separate injections of enough additional serum to make up the amount required for protection. The amount of virus used was generally 0·003 gm. per kilo body weight, and the total amount of immune serum 0·3 cc. per kilo body weight. This method has been tested in 15 patients whilst another was similarly protected by injecting the serum and virus separately.

By the titration in mice of the sera of vaccinated persons it was shown that the immunity rose to a height comparable to that following an attack of yellow fever, and remained there for at least six months. The virus could not be recovered from the blood of vaccinated persons or monkeys, except when the amount of immune serum was less than the minimum effective amount ; nevertheless, the immunizing reaction seems to be part of a true infectious process and is accompanied by a characteristic leucopenia. *E. H.*

MONTEIRO (J. Lemos) & TRAVASSOS (J.). Sobre a duração da actividade do antigeno para a reacção de fixação do complemento na febre amarella. [**Keeping Qualities of an Antigen for the Complement Fixation Reaction in Y. F.**]—*Brasil-Medico.* 1932. July 2. Vol. 46. No. 27. pp. 597–599. English summary.

" In a previous paper it was shown that the verification of both experimental yellow fever and immunity of persons having had the natural infection and living in endemic zones can be based on the complement fixation reaction. This reaction may take the place of the proof of protection of *rhesus* monkeys by means of immune serum, a process that is much more costly and tedious.

" The result of those previous experiments has recently been confirmed in regard to a few samples of sera received from Argentina for diagnosis, the antigen used having been prepared from the liver of infected *rhesus* nearly two years ago (21 months).

" Having thus retested several samples of sera previously examined, the authors found that the complement fixation power of their yellow fever antigen in the presence of immune sera has not changed for nearly two years and seems to have become somewhat strengthened." *H. H. S.*

STEFANOPOULO (G.) & CALLINICOS (G.). Absence d'anticorps pour le virus amaril dans le sang des sujets atteints de dengue. [**Absence of Y. F. Antibodies in Blood of Dengue Patients.**]—*C. R. Soc. Biol.* 1932. Aug. 12. Vol. 110. No. 28. pp. 1230–1231.

The authors made yellow fever protection tests in mice with the sera of eleven patients who had recovered from severe attacks of dengue in Greece. None of the sera gave any protection, whilst control mice inoculated with mixtures of the virus with either convalescent yellow fever serum or the serum of a horse immunized against this disease, were all protected. *E. H.*

WAKEMAN (A. Maurice) & MORRELL (Clare A.). **Chemistry and Metabolism in Experimental Yellow Fever in** *Macacus rhesus* **Monkeys. V. Acid-Base and Electrolyte Equilibrium.**—*Arch. Intern. Med.* 1932. May. Vol. 49. No. 5. pp. 826–835. With 3 charts in text. [15 refs.]

The concentration of proteins in the serum of monkeys was found to diminish during the course of infection with yellow fever, the reduction being mainly at the expense of the albumin fraction. In the early stage of the disease there was a rise in the globulins. Towards the end of the infection, serum bicarbonate is generally reduced, often considerably, the reduction being due chiefly to accumulation of acids, probably organic, and of inorganic phosphate. No characteristic alterations were found in the concentrations of base and chloride in the serum or in the urinary excretion of chloride. *E. H.*

HAAGEN (E.) & THEILER (M.). Untersuchungen ueber das Verhalten des Gelbfiebervirus in der Gewebekultur. Mit besonderer Berücksichtigung seiner Kultivierbarkeit. [**Behaviour of Y. F. Virus in Tissue Culture.**]—*Zent. f. Bakt.* I. Abt. Orig. 1932. Aug. 3. Vol. 125. No. 3/4. pp. 145–158. With 3 text figs. [25 refs.]

A more detailed account of the methods used by the authors [see this *Bulletin*, Vol. 29, p. 577]. The virulence of the virus in numerous passages in two serial cultivation experiments remained constant, from which it is evident that the methods which have been used for the cultivation of other viruses have also been successful in the case of yellow fever. A photograph is reproduced showing ganglion cells containing acidophile masses in the nuclei. *E. H.*

MARTIN-CHARPENEL (G.), MATHIS (Maurice) & KOLOCHINE (C.). Vérification d'une souche de virus amaril adaptée à la souris blanche. [**Verification of a Mouse Strain of Y. F.**]—*Bull. Soc. Path. Exot.* 1932. June 8. Vol. 25. No. 6. pp. 533–535.

A strain of yellow fever after eight passages in mice was still virulent for *Macacus rhesus*, in which it produced a typical and fatal infection. Mice inoculated intracerebrally with virus from this monkey died

after fairly uniform intervals, whilst the original strain before having been passaged in mice, required three passages in these animals before it became adapted and gave uniform results. *E. H.*

FROBISHER (Martin), Jr. Sobre a gordura nos figados de macacos mortos de febre amarella. [**The Liver Fat of Monkeys Dead of Y. F.**]—*Brasil-Medico.* 1932. Oct. 8. Vol. 46. No. 41. pp. 861–863.

States that the fat in the liver of a macaque dead of yellow fever differs from that of chloroform poisoning in the amount of iodine it will absorb. In other respects the results of analyses are similar in the two conditions; the findings are given in a table. *H. H. S.*

CAZANOVE (F.) & ROBERT (Léopold). Empoisonnement par le phosphore et fièvre jaune. [**Phosphorus Poisoning and Y. F.**]—*Bull. Soc. Path. Exot.* 1932. July 6. Vol. 25. No. 7. pp. 853–858.

A comparison of a case of phosphorus poisoning at Dakar with the analogous features noticed by early writers. *E. H.*

MONTEIRO (J. Lemos). Estudos sobre a febre amarella. Modernos conhecimentos sobre a infecção experimental. [**Studies on Y. F. Modern Knowledge of the Experimental Infection.**]—*Mem. Inst. de Butantan.* 1930. Vol. 5. pp. 53–170. With 44 graphs & 10 figs. on 5 plates. [222 refs.] English summary.

A good general account of the subject with special reference to work done at the Butantan Institute. *E. H.*

DINGER (J. E.). De beteekenis van het gele koortsvraagstuk voor Nederlandsch-Indië.—*Geneesk. Tijdschr. v. Nederl.-Indië.* 1932. Sept. 27. Vol. 72 No. 20. pp. 1331–1336.

SAWYER (Wilbur A.). Historia de la fiebre amarilla desde la epidemia de nueva Orleans en 1905.—*Bol. Oficina Sanitaria Panamericana.* 1932. July. Vol. 11. No. 7. pp. 689–699. [40 refs.]

RELAPSING FEVER AND OTHER SPIROCHAETOSES.

NICOLLE (Charles), ANDERSON (Charles) & LAIGRET (Jean). Etude des trois premiers cas de fièvre récurrente hispano-africaine observés en Tunisie et, plus spécialement, de leurs virus. [**Three First Cases of Spanish-African R. F. observed in Tunis, with Special Reference to the Virus.**]—*Arch. Inst. Pasteur de Tunis.* 1932. July. Vol. 21. No. 1. pp. 43–72. With 1 chart.

The authors have obtained spirochaetes belonging to the group *S. hispanica* de Buen, from three human cases of relapsing fever observed in Tunis. The transmitting agent in these cases was doubtless *Ornithodorus erraticus*, but specimens of this tick found at Carthage were infected with another strain, *S. erratici*, which seems to be more closely related to *S. normandi*, occurring in the wild rodents.

The spirochaetes of the third case of relapsing fever were transmitted experimentally by *O. erraticus*, *O. savignyi* and *O. turicata*, the latter from Mexico. In each species of tick the infection by bite was from one stage to the next. In one out of two experiments on the third case the spirochaetes persisted for six days in the louse, whilst as a general rule these organisms disappear from the gut within a day. Finally, the authors point out that the spirochaetes of the *hispanica* group are an excellent example in support of their view that these are not distinct species of relapsing fever spirochaetes, but only individuals that can be grouped together according to their pathogenicity and their method of transmission. *E. Hindle.*

DE LA CAMARA (Pedro), FERNANDEZ MARTINEZ (Jose), DE BUEN (Eliseo) & JUAREZ (Ernesto). Estudios sobre quimioterapia de la fiebre recurrente española. [**Chemotherapy of Spanish R. F.**]—*Medicina Países Cálidos.* Madrid. 1932. May. Vol. 5. No. 3. pp. 218–226. [14 refs.] French summaries.

The authors treated cases of Spanish relapsing fever with various arsenical preparations and from the results here recorded it would appear that the organism possesses a considerable degree of resistance to arsenicals, and to some more than others. Neosalvarsan and novarsenobillon were the most serviceable, but the number of patients treated by these was so small that no valid deductions can be drawn. Nine were given spirocid Bayer in doses up to 1 gm.; one had no fever after the third dose, but had 11 doses in all; in the other eight there was no result. Stovarsol and quiniostovarsol, though tried in several cases, were ineffectual; the fever continued even after prolonged courses of treatment with doses of 0·2 to 0·5 gm. *H. H. S.*

FELDT (Adolf). Ueber Arzneifestigung von Spirochäten im Tierversuch. [**Arsenic Resistance of Spirochaetes in Animal Experiments.**]—*Klin. Woch.* 1932. Aug. 13. Vol. 11. No. 33. pp. 1378–1380. [19 refs.]

The author has studied arsenic resistance in spirochaetes using strains of *S. recurrentis* in mice and *S. pallida* in rabbits. In mice strains of *S. recurrentis* were produced which were resistant against salvarsan and solganal respectively. It was found that the spirochaetes were especially sensitive to these drugs during some of the earlier passages after treatment. Thus a strain which at the beginning of the experiment was treated by intravenous injections of 1/1000 gm.

per cc. per 20 gm. mouse, in the next passage was resistant against 1/800, and in the 3rd against 1/500. Then it suddenly became more susceptible to the drug than the original normal strain and the 4th passage spirochaetes were killed by doses of 1/1000. At the 20th passage it was resistant against 1/200 and after a total of 40 passages reached its maximum resistance, against a dose of 1/100. Similar results were obtained in preparing strains resistant against solganal. This salvarsan-resistant strain, after passaging in normal mice for ten weeks lost its resistance, but the similar solganal-strain was still resistant after nineteen months. A strain of *S. pallida* after ten passages in rabbits during 14 months, was still resistant against double the minimum curative dose of salvarsan. *E. H.*

Hasskó (Alexander). **The Effect of Spirochete and Trypanosome Infection on the Distribution of Chemotherapeutical Drugs.**—*Jl. Comp. Path. & Therap.* 1932. Sept. Vol. 45. Pt. 3. pp. 230–239. With 4 figs.

An account of experiments with mice infected with *S. recurrentis* and subsequently treated with various substances. The results seem to show that the infection influences the function of the Kupffer cells of the liver and the perifollicular cells of the spleen, and hinders their phagocytic capacity at the culmination of the infection. The infection altered the deposition of drugs which are usually deposited in the reticulo-endothelial cells, and also the distribution of drugs such as neosalvarsan having affinities for the connective tissue. Infected mice injected intravenously with silver-salvarsan at the height of infection showed difficulty in breathing and did not move spontaneously whilst normal mice receiving similar doses showed no ill effects. The mice were killed 30 minutes after the injections and examined histologically, when a marked difference could be seen between the deposition of the drug in the normal and infected mice. In both the tissues of the lungs were uniformly impregnated with arsenobenzol granules, but in the Kupffer cells of the infected liver phagocyted granules were seen only in some places and were irregularly distributed, and they were absent from the phagocytic cells of the spleen and irregularly deposited through this organ. In the normal animal, on the contrary, the granules were evenly distributed in the Kupffer cells of the liver and in the phagocytic cells of the spleen. Infection with *Trypanosoma equiperdum*, which does not influence the function of the mesenchymal system, did not essentially alter the distribution of colloidal gold or salvarsan; therefore the effect of relapsing fever cannot be explained as being the result of saturation with phagocytosed parasites. The paralysing effect of relapsing fever on the reticulo-endothelial cells is only transitory and deposition may be observed two hours after the injection, although absent after half an hour. It is suggested that the delay may be explained by assuming that the drugs are first absorbed by the spirochaetes and become deposited only later together with them. *E. H.*

Kleine (F. K.) & Krause (Magdalene). Zur Uebertragung der Rückfallfieberspirochäten durch Zecken. [**Transmission of R. F. Spirochaetes by Ticks.**]—*Arch. f. Schiffs- u. Trop.-Hyg.* 1932. Nov. Vol. 36. No. 11. pp. 587–589.

The authors have fed uninfected *Ornithodorus moubata* received from Tanganyika on mice heavily infected with *S. duttoni*, and

subsequently on normal mice. After a varying number of feedings on mice, the ticks were finally killed and examined, some microscopically and others experimentally. It was found that the spirochaetes died out in some of the ticks [confirming previous observations on this subject by SCHUBERG and MANTEUFEL, and the reviewer]; in other instances the ticks might feed a number of times without producing any infection and yet later become infective; also ticks might produce infections and then fail to infect, and subsequently again become infective.

In opposition to the results of NICOLLE and his co-workers, adult ticks could be infected and become infective. The authors found no evidence in support of the view that spirochaetes as such are absent from infected ticks and were able to find the organisms not only in various organs of the body, but also in eggs laid by infected females.

E. H.

ADANT (M.). Au sujet d'un spirochète isolé chez un rat du Katanga et pathogène pour l'homme. [**A Spirochaete Isolated from a Katanga Rat and Pathogenic for Man.**]—*Ann. Soc. Belge de Méd. Trop.* 1932. Oct. 30. Vol. 12. No. 3. pp. 259–264.

The author has examined 51 examples of various species of wild rodents for relapsing fever spirochaetes by inoculation of blood and, in some instances, of the brain, into susceptible animals. The species examined included *Taterona liodon, Aethomys kaiseri* and *Mastomys coucha* (?), and were caught in various regions of Katanga Province. All these species are susceptible to experimental relapsing fever, and some of them were caught in localities where infected Ornithodorus occurred. Only one individual, an *Aethomys kaiseri* caught at Mukebo, was found to be naturally infected, the inoculation of its brain into another wild rat resulting in the appearance of spirochaetes in the blood of the latter after an incubation period of eleven days. This organism morphologically resembled the human relapsing fever spirochaete and when inoculated into two human subjects produced a single attack of fever with parasites in the blood. It also produced infections when inoculated into two species of monkeys and various wild rats, but the guineapig was refractory. *E. H.*

HIROKI (H.). Experimentelle Untersuchung ueber die Persistenz der Recurrensspirochaeten im Gehirn. I. Mitteilung: Ueber die grundlegenden Untersuchung mit dem mandschurischen Recurrensspirochaeten. [**Experimental Study of Persistence of R. F. Spirochaetes in the Brain. Part I. Experiments with Manchurian R. F.**]—*Jl. Oriental Med.* 1932. Aug. Vol. 17. No. 2. [In Japanese. German summary pp. 11–12.]

——. II. Mitteilung: Der Einfluss des künstlichen Verfahrens auf die Persistenz der Recurrensspirochaeten im Gehirn. [**Part II. Influence of Artificial Treatment on Persistence of R. F. Spirochaetes in the Brain.**]—*Ibid.* Sept. Vol. 17. No. 3. [In Japanese. German summary pp. 21–24.]

Using a strain of Manchurian relapsing fever maintained in rats and mice in the laboratory for ten years, the author infected 42 mice and examined the blood and brain for spirochaetes at various intervals. Both remained infective up to the 32nd day, after which the mice

were completely negative with the exception of a single individual that showed a residual brain infection 60 days after being inoculated, the blood being negative. In view of these results it is evident that persistent brain infections are very rare with this strain of relapsing fever and cannot be considered as an essential feature of the infection.

The author altered the virulence of the same strain of Manchurian relapsing fever in various ways in order to test PRIGGE and ROTHERMUNDT'S theory that residual brain infections are the result of low virulence and a feeble development of antibodies. The original strain had a mortality of 63 per cent. in mice ; after ten passages in mice using the percutaneous method of infection the mortality was 46 per cent. ; infecting *per os*, the mortality in mice was reduced to 49·2 per cent. after eleven passages, and to 21·1 per cent. after twenty-three passages ; and after five passages in man the mortality was 53 per cent. The original strain and the percutaneous one were compared for susceptibility to neosalvarsan but showed no difference. Mice were injected with three of the modified strains as well as the original one and subsequently examined for residual brain infections but all were negative. The immune serum of mice that had recovered from the strain of lowest virulence, protected mice not only against this strain, but also against the original one of much higher virulence. The author's results therefore give no support to PRIGGE and ROTHERMUNDT'S theories, but are in favour of the view that those strains of spirochaetes persisting in the brain do so as the result of a specific neurotropic character. *E. H.*

ROTHERMUNDT (M.). Untersuchungen ueber die Gehirnpersistenz der Rekurrensspirochäten bei weissen Mäusen und ihre Bedeutung bei der Beurteilung chemotherapeutischer Versuche. [**Persistence of R. F. Spirochaetes in Brains of White Mice ; its Significance in Chemotherapeutic Experiments.**]—*Ztschr. f. Immunitätsf. u. Experim. Therap.* 1932. Vol. 76. No. 3/4. pp. 201–213.

The author studied persistent brain infections in mice infected with various strains of *S. duttoni*, *S. crocidurae*, and *S. hispanica*, respectively. Tables are given showing the results of the examination of blood and brains in mice infected with a strain of *S. duttoni* in 1926, compared with a similar test of the same strain four years later. In 1926 all the mice showed residual brain infections, compared with only 30 per cent. in mice infected in 1930. The strain was then used for infecting a human patient, and afterwards inoculated into white mice, and in these the number showing residual brain infections rose from 30 to 55 per cent.

A strain of *S. crocidurae* gave 100 per cent. residual brain infections in 1928, but only 22 per cent. in 1931 ; and a strain of *S. hispanica* fell from 58 per cent. in 1928 to about 20 per cent. in 1931.

In view of these results the author urges that the examination of the brain for spirochaetes, as a criterion of the efficacy of any chemotherapeutic substance, and also the use of persistent brain infections for general chemotherapeutic experiments are of little value. The use of such strains only comes into question when it is desired to test whether or not any substance kills the spirochaetes in the brain.

E. H.

Mathis (C.) & Durieux (C.). Identité, à Dakar, de *Spirochaeta duttoni* var. *crocidurae* et du spirochète infectant dans la nature *Ornithodorus erraticus*. [**Identity of** *S. duttoni* **var.** *crocidurae* **with the Spirochaete found in** *O. erraticus* **at Dakar.**]—*Bull. Soc. Path. Exot.* 1932. July 6. Vol. 25. No. 7. pp. 843–853.

A detailed account of the cross immunity experiments showing the identity of strains of relapsing fever isolated respectively from *Ornithodorus erraticus*, caught wild at Dakar, from a human case of the disease (Boufflers) and from infected shrew-mice (*Crocidurus*) [see this *Bulletin*, Vol. 29, p. 555.] *E. H.*

Cannavò (L.). Ulteriore contributo allo studio della broncospirochetosi. [**Bronchial Spirochaetosis.**]—*Policlinico.* Sez. Prat. 1932. Mar. 7. Vol. 39. No. 10. pp. 363–368. With 4 text figs. [12 refs].

Except as a record of 5 more cases of bronchial spirochaetosis this article adds little to present-day knowledge of this condition, but it is a good summary of the views held, especially in Italy, regarding it. Four of the author's cases were chronic and one subacute, but all rapidly improved—cough was allayed, blood, if present, disappeared, spirochaetes diminished or disappeared—after a course of arsenobenzol intravenously. The characteristics are the chronicity with alternating amelioration and aggravation of symptoms, the want of correlation between symptoms and physical signs, and the slight constitutional disturbance.

The author concludes that bronchospirochaetosis may be endemic in Sicily and Southern Italy since many among those whom he has attended have never left the country, and that it is probably infective (though not highly) as he observed 3 cases in one family. [As usual no evidence, beyond mere association, is adduced that these spirochaetes are pathogens at all.] *H. H. S.*

Lorando (N. J.). Etude critique sur l'épidémie de l'île de Syra. Dengue et spirochétose.—*Bull. Soc. Path. Exot.* 1932. June 8. Vol. 25. No. 6. pp. 552–558. [12 refs.]

Remlinger, Cabanie & Bailly. Un caso de fiebre recurrente de espiroquetes virulentos para la cobaya en la zona española de Marruecos.—*Rev. Sanidad e Hig. Pública.* 1932. May. Vol. 7. No. 5. pp. 393–395.

Soriano (Francisco). Aportaciones al estudio de la fiebre recurrente española.—*Medicina Países Cálidos.* Madrid. 1932. July. Vol. 5. No. 4. pp. 303–309.

LEPTOSPIROSIS.

COPANARIS (Ph.). Quelques données nouvelles sur les infections à spirochètes du groupe ictérogène en Grèce. [**Spirochaetal Jaundice in Greece.**]—*Bull. Office Internat. d'Hyg. Publique.* 1932. Sept. Vol. 24. No. 9. pp. 1427–1430.

An account of an outbreak of 29 cases of spirochaetal jaundice in the Isle of Cephalonia. The symptoms suggested mild Weil's disease, but although the majority of the patients showed a subicteric colour from the fifth day, none had any haemorrhagic symptoms. Guinea-pigs were infected by intraperitoneal injections of the sediment of centrifuged urine.

The sera of nine of these patients were examined by SCHÜFFNER in Amsterdam and serologically were found to be distinct from Weil's disease, whilst the sera of three cases from the Isle of Syra were identical with them. It is evident that these outbreaks of spirochaetal jaundice are caused by more than one type of organism. *E. Hindle.*

PETZETAKIS (M.). A propos d'une épidémie de spirochétose ictéro-hémorragique à l'île de Syra. Origine hydrique de l'épidémie. Présence des spirochètes chez les rats d'égout, en Grèce. [**Epidemic of Spirochaetal Jaundice in the Island of Syra.**]—*Bull. Soc. Path. Exot.* 1932. May 11. Vol. 25. No. 5. pp. 411–416.

Further details on an epidemic of this disease in Syra [see this *Bulletin*, Vol. 29, p. 566]. A total of 31 cases with 3 deaths was observed, three-quarters of them occurring between 14th and 18th August, 1931. All the cases were traced to a particular café, which communicated with a filthy cellar, and the author considers that the water at this café may have been contaminated by infected rats. The nature of the spirochaete isolated was identified by the use of immune serum supplied by PETTIT.

The author also records the presence of *S. icterohaemorrhagiae* in the sewer rats of Syra and Athens. In Athens, 3 out of 96 rats showed spirochaetes in the kidney, and from one of these the infection was reproduced in the guineapig. *E. H.*

KIRSCHNER (L.). Waterspirochaeten en de ziekte van Weil. [**Water Spirochaetes and Weil's Disease.**]—*Geneesk. Tijdschr. v. Nederl.-Indië.* 1932. May 24. Vol. 72. No. 11. pp. 658–669. English summary.

Working in Bandoeng, the author attempted without success, to repeat the experiments made by BAERMANN and ZUELZER in Sumatra [see this *Bulletin*, Vol. 25, p. 602]. Using the same strain of water leptospira and the same breed of guineapig, it was found impossible to produce any signs of infection or persistence of the organisms in the blood. The Bandoeng house rat (*Rattus rattus diardi*) was found to be infected with *S. icterohaemorrhagiae*, four strains being isolated, all pathogenic for guineapigs. Spirochaetes were also cultured from three hot springs (38–42°C.) which had been used for years as a bathing establishment, but no cases of Weil's disease have yet been recorded in this region. *E. H.*

Thiry (U.) & van Meirhaeghe (A.). Recherches complémentaires sur les Leptospires aquicoles et les Leptospires ictéro-hémorragiques. [**The Water Leptospira and** *L. icterohaemorrhagiae.*]—*Rev. Belge Sci. Méd.* 1932. June–July. Vol. 4. No. 6. pp. 441–451. [Refs. in footnotes.]

The presence of coli-bacilli was not found to favour the development of these organisms in culture, and in many instances rendered the medium quite unfavourable to growth. Various strains of leptospira were grown in Verwoort's medium at varying degrees of pH; the pathogenic rat and human strains grew best at 7·4 to 8·6, in one instance up to 9·8, whilst the water strains grew at 5·8 to 7·4 or 7·8. A mouse strain grew at 7 to 7·8. *E. H.*

Kunstein (Erich). Ueber Verbreitung der Spirochäten vom Weiltyp in Freiburg i. Br. und Umgebung. [**Distribution of Weil-Type Spirochaetes in Freiburg i. Br. and its Environs.**]—*Ztschr. f. Immunitätsf. u. Experim. Therap.* 1932. Vol. 75. No. 1/2. pp. 173–190. [30 refs.]

The water leptospira was found in 60 per cent. of the waters examined including the drinking water of Freiburg, Ihringen and Lorrach. The pH of the infected waters varied between 6·9 and 7·5. Of 19 rats (*Mus decumanus*) from Freiburg i. Br., eight contained virulent *S. icterohaemorrhagiae* in their kidneys. *E. H.*

Dinger (J. E.). Het beloop der leptospirosis bij de Indische cavia. [**The Course of Leptospirosis in Guineapigs in Java.**]—*Geneesk. Tijdschr. v. Nederl.-Indië.* 1932. Oct. 11. Vol. 72. No. 21. pp. 1440–1448. With 3 figs. on 1 plate.

After experimental inoculation with leptospirae guineapigs in Java usually (in 2/3 of 69 cases) die within 5 days, as compared with 7–8 days in Europe. If material with diminished virulence is used a large percentage (2/3) of the animals tested continue to excrete virulent leptospirae in the urine for a considerable period.

In one case by means of such a carrier the author succeeded in producing a contact infection in the stable. This urges him to warn other workers to take heed against this possibility, which might introduce errors in experiments aiming to show alteration of water leptospirae into virulent organisms. *W. J. Bais.*

Sardjito. Verdwijning van de virulentie gepaard gaande met verandering van antigene eigenschappen van een ratten leptospira stam door te kweeken in water. [**Disappearance of Virulence together with Alteration of Antigenic Properties of a Rat Leptospira Strain by Cultivation in Water.**]—*Geneesk. Tijdschr. v. Nederl.-Indië.* 1932. Sept. 27. Vol. 72. No. 20. pp. 1364–1372. With 1 plate.

In view of the still unsettled question whether saprophytic water leptospirae may acquire pathogenic properties, the author studied this question by cultivating a rat leptospira strain in water. For the technical details of the methods by which a pure culture was obtained the original paper should be consulted. After five months the strain had lost the antigenic properties of the original one. At the same

time it had lost its pathogenicity for guineapigs which remained unaltered in the control, cultivated in ordinary serum dilution media. The morphological characteristics of the strain were slightly different from those of the original one ; it grew more luxuriantly and was more resistant to the oligodynamic influence of metals. It has in this way acquired the characteristics of a water leptospira. The author is inclined to believe that this alteration may be reversible. *W. J. Bais.*

Aulia & Cohen (A. J.) with Sardjito (M.) & Mochtar (A.). Een nieuwe spirillosis ? [**A New Spirillosis ?**]—*Geneesk. Tijdschr. v. Nederl.-Indië.* 1932. Sept. 27. Vol. 72. No. 20. pp. 1359–1363. With 4 figs. on 1 plate & 1 chart.

From the blood of a native suffering from a chronic intermittent and remittent fever, characterized by a relatively quick pulse, anaemia and haemorrhagic pericarditis, the authors cultivated a new spirillum for which they propose the name of *Spirillum cardiopyrogenes.* This spirillum is slightly pathogenic for guineapigs, but these animals can easily be infected by inoculation. The patient's serum contained antibodies against the strain. The organism is differentiated from *"Spirochaeta gallica"* (stated to have been found in trench fever) and from *S. morsus muris.* For details the original paper must be consulted. *W. J. Bais.*

Korthof (G.). Experimentelles Schlammfieber beim Menschen. [**Experimental Slime-Fever in Man.**]—*Zent. f. Bakt.* I. Abt. Orig. 1932. Sept. 21. Vol. 125. No. 7/8. pp. 429–434. With 1 fig.

Eleven patients were inoculated with cultures of leptospira from a strain of slime fever described by Dinger and Verschaffelt [see this *Bulletin*, Vol. 28, p. 314]. Two were resistant but the other nine became infected after an incubation period of 5 to 9 days. The organisms were observed by dark ground examination of blood that had been triple centrifuged ; and were recovered by culture of the blood in six out of seven patients and also by the inoculation of blood into mice and guineapigs. Serological tests showed that the infection was distinct from Weil's disease. It is considered possible that some of the cases described by Baermann and Zuelzer [see this *Bulletin*, Vol. 25, p. 602] may have been slime fever rather than Weil's disease.

E. H.

Caminopetros (Jean). Sensibilité du spermophile *Citillus citillus* à la spirochétose ictérohémorragique. [**The Susceptibility of the Spermophile to** *S. icterohaemorrhagiae.*]—*C. R. Acad. Sci.* 1932. May 30. Vol. 194. No. 22. pp. 1991–1992.

The author finds that this animal is even more susceptible than the guineapig to infection with *S. icterohaemorrhagiae.* Whatever the method of infection, or source of the spirochaete, the spermophiles invariably succumb to the disease, usually within 6 to 8 days, and the spirochaetes can be seen in the blood within 48 hours after inoculation. Jaundice is very distinct in the muscles and rib cartilages,

less evident in the skin and mucosa. The lungs show haemorrhages, the intestine is very congested, both spleen and liver are greatly enlarged. The infection is readily maintained in these animals by passage in series, and since the spirochaetes multiply in all the organs of the body, the spermophile would seem to be a more convenient host than the guineapig. *E. H.*

Petzetakis (M.), Melanidis (C.) & Tzortzakis. Sur la sensibilité du mouton, du chevreau et du porcelet au virus de la spirochétose ictérohémorragique par voie intratesticulaire. [**Susceptibility of Sheep, Kid and Young Pig to Intratesticular Inoculations of** *S. icterohaemorrhagiae.*]—*C. R. Soc. Biol.* 1932. June 17. Vol. 110. No. 21. pp. 482–484.

A lamb, a kid and a young pig were each inoculated intratesticularly with a suspension of the organs of a guineapig infected with *S. icterohaemorrhagiae*. Certain reactions were observed and spirochaetes were recovered from the blood of the lamb and the kid, up to seven days after inoculation in the case of the latter. The pig seemed to be more resistant. *E. H.*

Troisier (Jean). La spirochétose méningée expérimentale. [**Experimental Meningeal Spirochaetosis.**]—*Ann. Inst. Pasteur.* 1932. Sept. Vol. 49. No. 3. pp. 343–357. With 5 figs.

The subdural inoculation of a culture of *S. icterohaemorrhagiae* produced an acute or fatal meningitis in guineapigs and young rabbits, with or without jaundice. Young dogs showed two types of meningitis, one benign and curable, the other malign producing cachectic symptoms. In both types there was hypercytosis of the cerebrospinal fluid and an increase in the albumin content. Various monkeys were inoculated in the cerebrospinal canal and after a short incubation period showed fever and wasting, without jaundice, recalling human cases of the disease with meningeal symptoms. *E. H.*

Lissner (Lothar). Ein Fall Weil'scher Krankheit auf den Philippinen. [**A Case of Weil's Disease in the Philippines.**]—*Arch. f. Schiffs- u. Trop.-Hyg.* 1932. Nov. Vol. 36. No. 11. pp. 590–593. With 1 fig.

An account of a fatal case of jaundice clinically diagnosed as Weil's disease, although spirochaetes were not found. *E. H.*

Caminopetros (J.). L'évolution du spirochète ictérohémorragique dans l'organisme du rat.—*C. R. Soc. Biol.* 1932. July 19. Vol. 110. No. 25. pp. 905–906.

Laignel-Lavastine, Boquien (Y.) & Sterne (J.). Spirochétose ictéro-hémorragique compliquée de phlébite au décours de la maladie.—*Bull. et Mém. Soc. Méd. Hôpit. de Paris.* 1932. June 27. 48th Year. 3rd Ser. No. 22. pp. 1017–1020.

RAT-BITE FEVER.

Ruys (A. Charlotte). Het gedrag van *Spirillum minus* in de muis in verband met de diagnose der rattebeetziekte. [*Spirillum minus* **in the Mouse in Relation to the Diagnosis of Rat-Bite Fever.**]—*Nederl. Tijdschr. v. Geneesk.* 1932. July 9. Vol. 76. No. 28. pp. 3385–3388. With 1 fig. German summary.

In many cases the diagnosis of rat bite fever can be fully established from the history, the infiltration at the seat of the bite, the typical temperature curve, the rash and the result of administration of salvarsan. This diagnosis can be confirmed by dark-field examination for spirilla in the exudate obtained from the seat of bite or in the serous fluid obtained from a papule and, if this is not possible, by examination of Giemsa-stained smears. It is seldom possible to demonstrate spirilla in a thick blood film. If a number of relapses have occurred, probably the best examination to make is one for the presence of lytic antibodies. This reaction may be negative to begin with, but be positive with a relapse two months later. There still remains the animal test and here the guineapig is not very suitable as it is not a highly susceptible animal. The most susceptible of all test animals is the mouse. Unfortunately the mouse is itself liable to a spontaneous infection and the author goes in some detail into the characters which distinguish *Spirillum minus* var. *muris* from *Spirillum minus* var. *morsus muris*. Many observers consider them one and the same organism. But if the blood, and in addition the peritoneal fluid, of the mouse prove negative on a preliminary examination, then this animal may be considered suitable for a testing diagnostic inoculation. The inoculation of 0·2 cc. of the blood of the patient or of the inflammatory exudate from around the bite or from a papule of the exanthem will suffice. The first spirilla may be expected in the blood and peritoneal fluid of the mouse about a week later, and to reach large numbers and their maximum in 14 days, after which they disappear. *W. F. Harvey.*

Fróes (Heitor P.). Sodoku (Rat-Bite Disease) and its Incidence in Brazil. Brief Report of a New Case.—*Jl. Trop. Med. & Hyg.* 1932, June 15. Vol. 35. No. 12. pp. 185–186.

AMOEBIASIS AND DYSENTERY.

Amoebiasis.

Craig (Charles F.). **The Amebiasis Problem.**—*Jl. Amer. Med. Assoc.* 1932. May 7. Vol. 98. No. 19. pp. 1615–1620. [25 refs.]

The author concludes his survey by stressing the importance of treatment of infections with *E. histolytica* even though there may be no symptoms present that have attracted the attention of the infected individual. No one can tell what pathologic lesions may be present in such individuals or what lesions may develop in the future. Everyone harbouring this parasite should be treated as soon as the diagnosis is made. Treatment is for the best interests not only of the patient but also of the public, as the cure of every infection reduces by that much the danger of transmission of the parasite to others. *H. M. Hanschell.*

Craig (Charles F.). **The Pathology of Amebiasis in Carriers.**—*Amer. Jl. Trop. Med.* 1932. July. Vol. 12. No. 4. pp. 285–299. [31 refs.]

The author marshals evidence in support of his dictum that there is no such person as a healthy carrier of *Endamoeba histolytica*, if by that term is meant one in whom no lesions caused by the parasite are ever produced. If by the term " carrier " is meant an individual in whom the organism is living as a commensal without the production of lesions or symptoms due to its presence, it is very doubtful if the term should be used at all with reference to infection with this parasite. In every " carrier " cytolysis and necrosis of superficial epithelium continually occurs in microscopic areas of the intestine, followed fortunately in the majority of cases by rapid regeneration of epithelium and healing but in some definite ulceration occurs involving mucosa, submucosa, or muscular coats of intestine. *H. M. H.*

Hiyeda (Kentaro) & Suzuki (Michisaburo). **Pathological Studies of Human Amoebic Ulcers, especially those of Carriers.**—*Amer. Jl. Hyg.* 1932. May. Vol. 15. No. 3. pp. 809–823. With 6 figs. [10 refs.]

The authors describe and illustrate well the morbid histology of five human cases in which amoebic ulcers were found in the large intestine : two of these cases they consider to have been " carriers." The course of an amoebic infection appeared to be as follows. When the amoebae enter the large intestine, the epithelium produces a large amount of mucus which prevents many of the amoebae from entering the tissues. The struggle between amoebae and mucus may go on for 2 to 8 days (this the authors have experimentally verified). If the amoebae succeed in making contact with the gut epithelium they cause, by production of a toxin, necrosis or necrobiosis of the superficial epithelial layer. The amoebae, though actually in lumen of gut, multiply when they come in direct contact with the gut tissue. Eventually a roughness or erosion of the superficial layer of the gut mucous membrane occurs. The amoebae penetrate deeper into the intestinal wall, multiply there, and produce a large area of necrosis and a typical crater ulcer is formed. If bacterial infection be severe, an abscess develops, and in bursting forms a crater ulcer also, but

this is unusual. The amoebae within the tissue of the submucosa travel mostly in direction longitudinal to the gut axis; the ulcers are therefore wider below the surface; and there are formed funnel-like communications under the intact epithelial covering. If regenerating activities of epithelium and other tissue overcome the invading power of amoebae, reparation of the ulcer occurs causing thickening of submucosa. Restitutio ad integrum is never reached as long as amoebae are present. If small ulcers (such as the authors describe in the two "carrier" cases) occur with only a few amoebae, the balance of amoebic vitality and tissue resistance is well kept, and no marked intestinal symptoms occur. This is the state of the amoeba carrier. Cellular infiltration in amoebic ulcers mainly depends upon mixed infection of bacterial origin. None of the leucocytes has a special affinity for the amoebae or their products. *H. M. H.*

Hegner (Robert). **Studies on Amoebiasis in Panama.**—*Jl. Parasitology.* 1932. Mar. Vol. 18. No. 3. pp. 153–158. With 13 figs. on 2 plates.

An interesting and valuable contribution, well illustrated. The author's experimental studies have shown that there is a mechanical penetration of the gut mucosa by the amoebae in monkeys experimentally infected; that immature cysts develop in faeces outside the body of the host; that excystation occurs in the small intestine of monkeys within about 3 hours after being inoculated into the stomach; that a single organism emerges from the cyst; and that cysts with one or two nuclei excyst as well as those with four nuclei. He finds no ground for the view that amoebae in gut lumen originate in the tissues; on the contrary they arise by multiplication in the lumen. The origin of amoebic ulcers is not yet clear. Bacteria probably play a role and follicular ulcers of bacterial origin may be involved, but amoebae appear unquestionably to be the active agents in formation and growth of these ulcers. *H. M. H.*

Meleney (Henry E.), Bishop (E. L.) & Leathers (W. S.). **Investigations of *Endamoeba histolytica* and Other Intestinal Protozoa in Tennessee: III. A State-Wide Survey of the Intestinal Protozoa of Man.**—*Amer. Jl. Hyg.* 1932. Sept. Vol. 16. No. 2. pp. 523–539. With 7 maps & 2 graphs. [12 refs.]

20,237 persons in the rural portions of 69 counties of Tennessee were examined for intestinal protozoa. Four contiguous counties on the eastern Highland Rim had the highest incidence of *E. histolytica* in the State, with an average incidence of 22·8 per cent. on one examination. The age incidence for each protozoon followed the curve usually described for it. The amoebae increased rapidly in incidence during childhood, showed a further slight increase in middle adult life and decreased after the age of sixty. *Giardia* reached its peak of incidence in early childhood and fell to a low level in adults. *Chilomastix* showed a peak in early childhood, decreased in the adolescent period and rose again in adults. Incidence of amoebae in whites and negroes showed no significant differences. *Chilomastix* and *Giardia* were found somewhat more prevalent in whites than in negroes. Habits of the people as influenced by physiographic environment probably influence the incidence of *E. histolytica* in the rural population groups studied. *H. M. H.*

i. Izar (Guido). Amebiasi infantile. [**Amoebiasis in Infants.**]—*Riforma Med.* 1932. Sept. 17. Vol. 48. No. 38. pp. 1435–1436.
ii. Manson-Bahr (Philip). **Amoebic Dysentery in Infancy.** [Correspondence.]—*Lancet.* 1932. Dec. 10. p. 1304.

i. The author states that amoebic infection is more common in infants than is generally believed; even those at the breast and in the early days of life are not immune. He quotes Tarro [? in Messina; the reference is not given] as recording the ages of 30 children dying of amoebiasis, on whom post mortem examinations were carried out; they were 8, 16, 21, 23 days, 1–2 months (6 cases), 2–3 months (4), 3–4 months (4), 4–5 months (2), 5–6 months (1), 6–7, 7–8, and at 9 months (2 each) and between 1 and 2 years (3).

Clinically, some children present typical dysenteric symptoms, others suffer from algid, choleraic symptoms, or those of sepsis and toxaemia, or a dystrophic or "gastrodyspeptic" syndrome. In a majority of instances infection is said to be contracted from the custom of the mothers having their children sleeping with them in bed.

Treatment is difficult because children so young as these do not tolerate emetine well. Thus, the doses which he uses (and even these with great caution) are: Up to 1 year a total of 5 mgm. in 10 days; from 1–6 years 7·5 mgm., from 6–10 years 10 mgm. He prefers stovarsol of which he gives: Up to 1 year a daily dose of 3 mgm. for a fortnight; 1–6 years 6 mgm.; 6–10 years 10 mgm.

ii. Dr. Manson-Bahr comments upon Dr. Izar's findings and believes that the cases described by the latter were more probably infections of bacillary dysentery. He bases this conclusion on the ground that ulcers do not develop for a year, possibly longer, after oral infection with the Entamoeba. Dr. Manson-Bahr himself, in a long experience with this disease, has never met with a patient under the age of 5 years. He notes also that the large macrophages, abundant in the stools of bacillary dysentery, closely simulate, and are often mistaken for, *E. histolytica*. *H. H. S.*

Alexeieff (A.). Sur la "triade intestinale" dans la dysenterie amibienne. [**The Intestinal Triad in Amoebic Dysentery.**]—*Arch. f. Schiffs- u. Trop.-Hyg.* 1932. Aug. Vol. 36. No. 8. pp. 440–444. With 2 text figs.

Manson-Bahr in Fiji (1910) first noted the characteristic cellular exudate and its importance in differential diagnosis in bacillary and amoebic dysentery respectively. This exudate was further and more intensively studied in the near East during the Great War by the late Dr. John Anderson and many authors since have given attention to it and reported on its value in diagnosis among whom Dr. Alexeieff's observations deservedly rank high. In this paper (well illustrated) he pays tribute to Anderson's work and confirms his observations. It is often hard to find *E. histolytica* in the stools of early amoebic dysentery: the diagnosis is certain however if pseudopyknotic nuclei of polymorphonuclear leucocytes (Anderson's pyknotic) are present and is finally confirmed if eosinophils and Charcot-Leyden crystals be found as well. These latter however appear later in the illness. He cites clinical observations in support of the contention that the number of these polymorphs with pseudo-pyknotic nuclei depend directly on the depth to which infection has penetrated. They are most numerous in submucous invasions, least in superficial invasions. In the latter the intestinal juice dilutes the ferments of the amoeba.

With ANDERSON he believes that the amoebae secrete a proteolytic ferment which digests the cytoplasm but has less action on the nuclei of the polymorphonuclear leucocyte. ANDERSON reported 83 per cent. of pyknotic polymorphs in his cases. Dr. Alexeieff cites one of his cases—a severe one—with 85 per cent. He insists that the presence of pseudopyknotic nuclei is the first recognizable sign of infection of the gut by *E. histolytica*. *H. M. H.*

MACKIE (Thomas T.). **Iodoxyquinolin Sulphonic Acid (Anayodin) in the Treatment of Intestinal Amoebiasis.**—*Twentieth Ann. Rep. Med. Dept. United Fruit Company, Boston, Mass.* 1931. pp. 112–122. [16 refs.]

The drug was tried on seven acute and chronic cases of amoebic dysentery, and 23 chronic cyst passers. In both groups it eliminated promptly *E. histolytica* from the stools. Seventeen cases re-examined from 10 days to 2 months after treatment disclosed no evidence of amoebic infection. The drug appeared to be relatively ineffective against other intestinal protozoa. *H. M. H.*

GAIDE & NIEL. Note sur la recrudescence des abcès du foie au Tonkin et considérations sur le traitement émétinien de l'amibiase. [**Recrudescence of Liver Abscess in Tonking. Treatment by Emetine.**]—*Bull. Soc. Méd.-Chirurg. Indochine*, 1932. Apr. Vol. 10. No. 3. pp. 347–365. With 1 chart.

Between 1918 and 1932 102 cases of liver abscess occurred with 27 deaths. The author gives a critical survey of these cases and in conclusion urges the necessity of watching closely all patients suffering from hepatic congestion and amoebic dysentery, of energetic emetine treatment from the start, and of continuing after the first symptoms have disappeared, provided that more than 1 gram be not given in any one month, and of systematic exploratory puncture of liver.

H. M. H.

BIGGAM (A. G.). **A Consideration of Certain Points in the Treatment of Amoebic Abscess.**—*Jl. Roy. Army Med. Corps.* 1932. Aug. Vol. 59. No. 2. pp. 141–144. With 1 fig.

The author describes two cases of dysentery in whom sigmoidoscopy revealed characteristic ulceration of bowel, active *E. histolytica* being demonstrated in the stools. On admission to hospital neither patient had any sign or symptom of liver involvement, and both showed satisfactory improvement in bowel symptoms, Case 1 on yatren enema and oral stovarsol, and Case 2 on emetine bismuth iodine and yatren enema ; both however developed amoebic hepatic abscess. An experience (notes the author) compelling great caution in accepting claims for the curative value in amoebic abscess of liver, or in early amoebic hepatitis, of any drug other than emetine. *H. M. H.*

NORTIER (A.). Over de therapie van lever-amoebiasis. [**Treatment of Amoebiasis of the Liver.**]—*Geneesk. Tijdschr. v. Nederl.-Indië.* 1932. Aug. 30. Vol. 72. No. 18. pp. 1184–1191. With 4 charts. [11 refs.]

The author pleads for intrahepatic emetine injections (in the abscess cavity) in cases of large abscess of the liver. Five illustrative case

reports are quoted. The author's arguments are chiefly: (1) The danger of secondary infection in cases of surgical drainage; (2) the way in which the abscess contents act as a medium to carry the drug to all the corners of the cavity. As a rule 90 mgm. of emetine is injected daily into the abscess, and subcutaneous injections are given also. *W. J. Bais.*

KATAROPULO (A.). Amöbenperikarditis. [**Amoebic Pericarditis.**]—*Arch. f. Schiffs- u. Trop.-Hyg.* 1932. Oct. Vol. 36. No. 10. pp. 544–548. [12 refs.]

Describes and discusses, with references to other authors, a case of liver abscess with dysenteric symptoms, ten years after a severe dysentery. The liver pus revealed *E. histolytica*. 7 years later liver abscess recurred and ruptured into the pericardium setting up fibrinous pericarditis; death. In the liver abscess pus living *E. histolytica* were found, none in the pericardial contents and many in the pericardial walls. *H. M. H.*

PENSO (Giuseppe). L'"Entamoeba vesicalis" (Penso). [**Entamoeba vesicalis.**]—*Ann. di Med. Nav. e Colon.* 1932. July–Aug. 38th Year. Vol. 2. No. 1–2. pp. 413–414. With 4 figs. on 1 plate.

In an earlier paper reviewed in this *Bulletin* (Vol. 28, p. 263) the author gave the name *Entamoeba vesicalis* to certain structures which he had seen in urine and had regarded as amoebae. The reviewer came to the conclusion that the author was not dealing with amoebae but with degenerate cells. In the present paper the author returns to the subject and describes a further case in which he claims that the same amoeba was found. He also mentions three other workers who have stated recently that they had found this amoeba in human urine. The author in support of his contention that he is dealing with an amoeba gives four microphotographs illustrating four phases in the production of a pseudopodium by an amoeba. The pseudopodium is shown as a perfectly spherical structure which in the fourth illustration is attached to the amoeba by a narrow portion and has reached a size greater than the amoeba itself which, moreover, has shown no diminution in size or alteration as regards its shape or the position of its nucleus. The supposed pseudopodium is evidently of slow formation as four microphotographs were taken. What it is cannot be stated definitely. It may be a product of cell degeneration or of some change in the medium but it is certainly unlike any pseudopodium ever seen by the reviewer who finds nothing in the present paper to justify him in changing the opinion he originally expressed. As regards the other observers mentioned the author says that confirmation of his observations are beginning to appear. Two of these have recorded single cases while one states that he has found *E. vesicalis* in eleven cases of cystitis in Somaliland. It seems clear in view of the author's claims that these statements cannot be accepted without confirmation. *C. M. Wenyon.*

CROPPER (C. F. J.) & MITRA (Jagdish). **A Case with *Entamoeba histolytica* in the Urine.**—*Indian Med. Gaz.* 1932. July. Vol. 67. No. 7. pp. 387–388.

The authors identified this *Entamoeba histolytica* on the following reported data. On one occasion in one preparation of urine they saw "an amoeba [sic] exhibiting movements typical [sic] of *E. histolytica*; its diameter was about 20μ. It contained 4 red cells. No clear differentiation between ectoplasm and endoplasm could be made out." *H. M. H.*

Broc (René). Un cas d'amibiase pulmonaire pure chez un musulman tunisien. Congestion pulmonaire avec hémoptysie. Guérison rapide par l'émétine. [**Pulmonary Amoebiasis in a Tunisian.**]—*Tunisie Méd.* 1932. Jan. Vol. 26. No. 1. pp. 16–32. With 1 chart in text & 2 figs. on 1 plate. [22 refs.]

Pulmonary symptoms. Sputum examinations revealed nothing definite but precystic amoebae were found in the stools. All pulmonary symptoms rapidly disappeared after emetine injections. [So much and no more for the diagnosis " d'amibiase pulmonaire pure." !] *H. M. H.*

Koch (Dorothy A.) & Reed (Alfred C.). *Endamoeba histolytica.* **Unusual Strains.**—*Amer. Jl. Trop. Med.* 1932. July. Vol. 12. No. 4. pp. 307–316. With 10 figs. on 2 plates.

An interesting study, well illustrated, of the amoebae found in a case of dysentery, from which the authors conclude that they were here dealing, in one patient, with two atypical strains of *E. histolytica* : the first large, highly pathogenic and rapidly eradicated by carbarsone ; the second smaller, also somewhat atypical in the trophozoite form, but forming cysts, and therefore probably more resistant to treatment.

H. M. H.

Kiefer (Everett D.). **The Craig Complement-Fixation Test for Amebiasis in Chronic Ulcerative Colitis.**—*Amer. Jl. Med. Sci.* 1932. May. Vol. 183. No. 5. pp. 624–631. [12 refs.]

Case histories of 19 cases of chronic ulcerative colitis are briefly reported. The author points out and discusses the great difficulty in differentiation between chronic ulcerative colitis and chronic amoebic colitis. Of the 19 cases 15 gave a positive result with the Craig complement fixation test for *E. histolytica* infection. The author draws no conclusions as to the significance of this finding, but suggests that chronic ulcerative colitis may be a pyogenic infection of the colon superimposed upon an original amoebic ulceration. *H. M. H.*

i. Heathman (Lucy). **Studies of the Antigenic Properties of Some Free Living and Pathogenic Amebas.**—*Amer. Jl. Hyg.* 1932. July. Vol. 16. No. 1. pp. 97–123. With 1 fig. [35 refs.]

ii. Sherwood (Noble P.) & Heathman (Lucy). **Further Studies on the Antigenic Properties of Pathogenic and Free Living Amebas. II. Complement Fixation in Amebic Dysentery.**—*Ibid.* pp. 124–136. [16 refs.]

i. The author's work leads her to conclude that this taxonomic study by means of the antigenic properties of amoebae corroborates Schaeffer's classification based on morphology, in the sense of being able to distinguish in a clear cut way between species as classified by Schaeffer.* It had not been possible always to place them in the same order by serological methods as by morphological ones. Both free living and pathogenic amoebae appeared to be good antigens as judged by formation of precipitin and complement-fixation antibodies. The precipitate was characteristically light but definite. It was possible in only a few cases to infect young kittens with supposedly pathogenic strains of *E. histolytica.* It was impossible to infect kittens

* Taxonomy of Amebas, *Carnegie Inst. Washington Publ. 345.* 1926.

with the free living *Flabellula citata*, although it possessed other properties similar to *E. histolytica* ; *e.g.*, it readily ingested and digested red blood cells from different animal species. Ingestion and digestion of red blood cells is not therefore a suitable criterion for pathogenicity of amoebae.

ii. The authors set out to repeat and investigate CRAIG'S previous work. They found that using 3 new amoeba lipoid antigens and the Kolmer haemolytic technique positive complement fixation (C. F.) in amoebic dysentery occurred in nearly 100 per cent. of the cases (preparation of antigens and technique here described). Four out of the 111 individuals used for controls gave four plus C. F. with one (most sensitive) of the antigens, and negative results with the other two. Two of these four had been handling cultures of *E. histolytica* for several months but the other two gave no history of contact or infection and stool examinations of all four were negative. In both infected and apparently normal persons, C. F. when it occurs is due to presence of specific immune bodies, specific for an amoeba lipo-protein or lipoid (haptene), which is an expression of natural immunity or latent infection ; and not necessarily of tissue immunity. The occurrence of positive C. F. in some cases of chronic ulcerative colitis [see KIEFER, above p. 25] in which no amoebae were found needed further investigation. *H. M. H.*

MENENDEZ (Paris E.). **Serological Relationships of *Entamoeba histolytica*.**—*Amer. Jl. Hyg.* 1932. May. Vol. 15. No. 3. pp. 785–808. [22 refs.]

The medium described by TANABE and CHIBA [this *Bulletin*, Vol. 29, p. 124] was found to be best because of the relatively poor growth of bacteria associated with *E. histolytica* under cultivation ; partial anaerobiasis was of value in maintaining viability of stock *E. histolytica* cultures ; standardization of bacteria accompanying cultures of the amoeba facilitated control of non-specific serological reactions. Specific complement binding, precipitin and amoebolytic antibodies were demonstrable in the sera of rabbits immunized with *E. histolytica* and *E. barreti*. Negative benzoin-flocculation tests were obtained with *E. histolytica* immune sera. Rabbits immunized with *E. histolytica* and *E. barreti* were found to be specifically hypersensitive to the corresponding antigens three months after the last injection. Any strain of *E. histolytica* gave effective antigen for use in diagnosis of infections with that parasite.

The author concludes that results of his study not only indicate the practical application of immunological tests in diagnosis of amoebic infections but also their usefulness in classification of certain animal parasites. *H. M. H.*

BACILLARY DYSENTERY

MACKIE (Thomas T.). **Ulcerative Colitis due to Chronic Infection with Flexner-Y Bacillus. Report of Case with Cure by Autogenous Vaccine.**—*Jl. Amer. Med. Assoc.* 1932. May 14. Vol. 98. No. 20. pp. 1706–1710. [28 refs.]

A valuable study. The author notes that the Flexner-Y group of dysentery bacilli may cause sporadic cases of mild diarrhoea and

dysentery. Occurrence of these cases does not require presence of epidemic conditions. A significant number of these individuals may become persistent carriers and develop ulcerative lesions of the lower part of the bowel, even though the initial acute attack may have been very mild. The lesions of chronic bacillary dysentery differ in no respect from those encountered in many of the cases of so-called ulcerative colitis. Intermittence of the excretion of the bacilli is one of the characteristics of chronic Flexner bacillus infection, and repeated cultures over a period of weeks may be necessary to demonstrate them. Taking cultures direct from the floor of the ulcer through the sigmoidoscope materially increases probability of recovering the organism. Autogenous vaccine in adequate dosage sufficiently prolonged is an efficient therapeutic procedure. *H. M. H.*

Browne (Donovan C.). **Further Study on the Incidence of** *B. dysenteriae* **in the Normal Individual.**—*Proc. Soc. Experim. Biol. & Med.* 1932. June. Vol. 29. No. 9. pp. 1272–1273.

The author had previously reported finding in stools of 50 healthy individuals selected from New Orleans public clinics *Bact. dysenteriae* in 6 cases, one a true Shiga type and five the lactose fermenting strain described by Duval. He now reports results of a similar investigation of 50 healthy individuals selected from private practice and living under much better hygienic conditions. In 3 cases *Bact. dysenteriae* was found, one Duval, one Hiss Y and one Flexner. *H. M. H.*

Wats (R. C.) & Jafar (M.). **Dysentery in Burma (Military Stations) with a Note on Some Post-Dysenteric Infections.**—*Indian Med. Gaz.* 1932. Aug. Vol. 67. No. 8. pp. 446-448. With 1 chart.

Total number of cases with mucus or blood and mucus in stools 269. *B. dysenteriae* isolated in 126. *E. histolytica* in 34. Bacillary exudate but no dysentery bacilli or pathogenic amoebae found 46. Indefinite exudate 106. Cases showing *Bact. dysenteriae*, Flexner 102; Shiga 11; Schmitz 8; Sonne 3; Morgan No. 1, 1; Carolinus 1.

H. M. H.

Iguchi (Jokai) & Otsubo (Goya). **Oral Vaccination for the Prevention of Dysentery and** *Ekiri*.—*Jl. Public Health Assoc. Japan.* 1932. May. Vol. 8. No. 5. pp. 4–9.

——, Ohotsubo (Itsuya), Sugina (Tameji), Kanno (Jiro), Nagatsuka (Takashima), Okuaki (Giichi), Kashiwagi (Seisho), Oshima (Kinko), Ehuchi (Churoku), Hachiya (Tsunetaro), Misaka (Osuke) & Ohsuka (Takeshi). **Prophylaxis of Dysentery by** *per os* **Immunization.**—*Kitasato Arch. Experim. Med.* 1932. July. Vol. 9. No. 3. pp. 218–223.

Observations during two consecutive years on 130,000 children, two to six years old, in the Tokyo district, who were made to take a dysentery bacillus vaccine per os, showed a reduction by half of the morbidity rate of dysentery and *Ekiri*, and some lowering of the death rate. The authors are arranging to make another experiment on some 470,000 children. Results to be reported in due time. *H. M. H.*

VAN DER WALLE (N.). Over het conserveeren van dysenterische ontlasting. [**On the Preservation of Dysenteric Stools.**]—*Geneesk. Tijdschr. v. Nederl.-Indië.* 1932. Aug. 2. Vol. 72. No. 16. pp. 1053–1056.

The author's aim was to preserve dysenteric stools so as to make successful bacteriological examination possible after an interval of a few days. Two methods were applied :—

(1) Flakes of the mucus were washed in salt solution, put in a watch glass and exsiccated in the dark above calcium chloride.

(2) Mucus flakes were enclosed in paraffin of a low melting point (45°C.).

In both cases after the lapse of time desired the material is examined again, in the second case after melting the paraffin, shaking it with culture broth and keeping it in the incubator for a few hours. Endo- and bile-Endo-plates were used.

After keeping the specimen for 6–14 days at room temperature the author succeeded in cases which primarily had given positive results in obtaining such results again by the first method in 11 out of 14 cases, by the second in 13 out of 31 of Shiga-Kruse bacillus infection and in 4 out of 4 and in 2 out of 6 cases of infection with pseudo-dysentery bacillus.

The results of the first method are apparently by far the better ; yet the second (paraffin) method is more practical. The method may be of special importance where certainty is called for about the character of dysentery epidemics in places far away from the laboratory. In such epidemics the bacteriological confirmation even of one or two cases is of paramount importance. The author actually succeeded in obtaining positive results in this way in 4 samples sent to the laboratory from long distances and kept for 5–14 days (3 samples were negative). *W. J. Bais.*

AMEDEO (Mattei). Su di alcuni casi di cistite amebica da " E. vesicalis."—*Ann. di Med. Nav. e Colon.* 1932. Jan.–Feb. 38th Year. Vol. 1. No. 1/2. pp. 24–47. With 6 figs. on 1 plate.

ANDERSON (Hamilton H.). Current Medical Problems of Central America, with Special Reference to the Treatment of Amebiasis.—*Southwestern Med.* 1932. Mar. Vol. 16. No. 3. pp. 110–113.

CANNAVO (L.). Su di alcuni aspetti clinici dell'epatite amebica.—*Riforma Med.* 1932. Mar. 26. Vol. 48. No. 13. pp. 471–478. [13 refs.]

GAGLIO (Vito). Sull' amebiasi nei suoi rapporti con la chirurgia.—*Riv. Sanitaria Siciliana.* 1932. Apr. 1 & 15. Vol. 20. Nos. 7 & 8. pp. 468–470, 473–477 ; 522–524, 527–534, 537–540. English summary (9 lines).

GRIFFIN (M. F. N.). The Death of an Entamoeba.—*Jl. Roy. Army Med. Corps.* 1932. May. Vol. 58. No. 5. pp. 371–372.

HÉRIVAUX (A.), RAKOTOSON (J.) & RANOARIVONY (E.). A propos d'un cas de dysenterie bacillaire dans le Sud-Ouest de Madagascar.—*Bull. Soc. Path. Exot.* 1932. Apr. 13. Vol. 25. No. 4. pp. 374–375.

HIYEDA (K.). Intestinal Amoebiasis.—*Jl. Oriental Med.* 1932. Sept. Vol. 17. No. 3. [In Japanese. German summary pp. 17–20.]

HOLL (Gustav). Ueber einen atypischen Fall von Amöbenruhr.—*Arch. f. Schiffs- u. Trop.-Hyg.* 1932. Sept. Vol. 36. No. 9. pp. 500–502. With 1 fig.

HOOD (Alexander). Dysentery.—*Jl. Roy. Army Med. Corps.* 1932. May. Vol. 58. No. 5. pp. 350–360.

Lama (Angelo). Epatite con incipiente ascesso e pregressa appendicite da amebiasi.—*Arch. Ital. Sci. Med. Colon.* 1932. July 1. Vol. 13. No. 7. pp. 426–433. English summary (2 lines).

Karczag (L.). Ueber das Striasyndrom bei Dysenterie.—*Deut. Med. Woch.* 1932. Feb. 12. Vol. 58. No. 7. pp. 249–250. [6 refs.]

Klein (S. A.). Een geval van balantidiosis bij den mensch.—*Nederl. Tijdschr. v. Geneesk.* 1932. Oct. 29. Vol. 76. No. 44. pp. 5050–5057. With 4 figs. on 1 plate. English summary (3 lines). [13 refs.]

Lawrence-Smith (A. M.). Unsuspected Amoebic Dysentery with Hepatitis.—*Jl. Roy. Nav. Med. Serv.* 1932. Apr. Vol. 18. No. 2. pp. 135–136. With 1 chart in text.

Moretti (Agostino). Contributo alla conoscenza delle coliti da "Balantidium coli."—*Policlinico.* Sez. Prat. 1932. June 13. Vol. 39. No. 24. pp. 922–925.

Moschini (S.). Contributo allo studio della cistite amebica nell'infanzia.—Reprinted from *Riv. di Clin. Pediatrica.* 1932. Vol. 30. No. 5. 16 pp. [54 refs.]

Nandi (P.). Treatment of Bacillary Dysentery.—*Calcutta Med. Jl.* 1932. Mar. Vol. 26. No. 9. pp. 368–382.

Panayotatou (Angelique). Sur un cas de "grippe" compliqué d'"amibiase."—*Arch. Ital. Sci. Med. Colon.* 1932. July 1. Vol. 13. No. 7. pp. 390–392.

Reed (Alfred C.) & Johnstone (Herbert G.). Method for Diagnosis of Amebiasis.—*Jl. Amer. Med. Assoc.* 1932. Aug. 27. Vol. 99. No. 9. p. 729.

St. John (J. H.). A New Medium for the Cultivation of *Endamoeba histolytica.*—*Amer. Jl. Trop. Med.* 1932. July. Vol. 12. No. 4. pp. 301–305. With 1 fig.

Ségal (J.). Amibiase et anémie pernicieuse.—*Rev. Méd. et Hyg. Trop.* 1932. May–June. Vol. 24. No. 3. pp. 154–157.

Skinner (A. H.) & Gillison (K.). Acute Pericardial Effusion simulating Hepatic Abscess.—*Chinese Med. Jl.* 1932. May. Vol. 46. No. 5. pp. 465–472. With 1 chart in text.

Snijders (E. P.). Over het leverabsces in de Tropen.—*Nederl. Tijdschr. v. Jeneesk.* 1932. Mar. 5. Vol. 76. No. 10. pp. 1066–1080. With 3 text figs.

Spangenberg (Juan Jacobo). Sobre un caso de hepatitis amebiana dando un sindrome de oclusión intestinal a repetición.—*Arch. Argentinos de Enferm. Aparato Digest. y Nutric.* 1931. Dec. Vol. 7. No. 2. pp. 165–184. With 8 text figs.

Vialatte (E. F. C.) & Remontet (J. E.). Contribution à l'étude clinique de l'hépatite amibienne congestive.—*Arch. Méd. et Pharm. Milit.* 1932. Apr. Vol. 96. No. 4. pp. 381–409. With 7 charts. [14 refs.]

Woodcock (H. M.). Proteolytic Ferment in Amoebic Dysentery. [Correspondence.]—*Lancet.* 1932. Oct. 29. p. 972.

MISCELLANEOUS.

United Fruit Company, Boston, Mass. Twentieth Annual Report. Medical Department. 1931. Section I. pp. 3–33.— **Comments on Some of the More Important Diseases occurring in the Tropical Divisions.**

The extent and scope of the work of this medical department is seen from the following data—The employees in 1931 numbered 73,251 of whom 65,680 were located in the tropical divisions with 76,640 non-employees (families of employees and others). The personnel of the medical department includes 45 physicians and laboratory technicians [the list of staff shows 51 with medical qualifications]. 22,752 patients were treated in the 10 hospitals and the average number daily was 724. The average treated daily at dispensaries was 1,027.

A table of causes of morbidity and mortality gives for hospital cases (morbidity), malaria (4,918), diseases of digestive system, influenza, injuries, venereal diseases, in that order ; and for dispensary cases similar frequencies, with ulcers a prominent feature. For deaths pneumonia is easily first, followed by malaria, heart disease, tuberculosis, diarrhoea, nephritis. Notes follow of the more prevalent diseases. Of venereal diseases it is said that they " probably rank next to malaria as a cause of lowered vitality and resistance in the tropical countries where our plantations are located. Their enervating and debilitating effects undoubtedly are as prevalent and more destructive of human energy and life than those of hookworm." A small epidemic of infectious jaundice in Colombia is discussed at some length ; in one of the first cases death resulted with symptoms suggestive of yellow fever. In 1930 and 1931, 58 cases, all natives, were admitted to the Santa Marta hospital with 8 deaths ; no foreigners have been affected which, together with the absence of epidemic, is against yellow fever. Investigation is being continued.

The bulk of the " comments " concern malaria.

" Despite the most strenuous efforts and the utilization of all known means of anti-malarial measures, the incidence of malaria can be reduced only to proportions which do not materially handicap the individual or the community ; but total eradication of the disease seems to be almost an impossible accomplishment with the present knowledge and conditions."

Table G (p. 31) shows the ratio of aestivo-autumnal infections to tertian infections from year to year in two divisions ; the author cannot offer any satisfactory explanation for the fluctuations in the relative proportions ; the rainfall, he states, is not a determining factor. The number of employees per thousand per annum admitted to hospital for malaria has fallen from over 200 in 1925 and 1926 to 85 and 56 in 1930 and 1931. It is calculated that the anti-malaria work has resulted in 6,168 less cases of malaria among employees, 7,738 less hospital admissions and 107,283 less days in hospital than there would have been if the rates had continued the same.

Some antimalarial measures are not easily carried through when " not infrequently, physical violence is threatened when sanitary inspectors, farm foremen and other employees in charge of labor units, endeavor to persuade laborers and members of their families to take the daily doses of quinine and plasmochin for a sufficiently extended period to effect a cure and to render them non-infectible to mosquitoes."

The antimalarial measures employed are described. Briefly they consist of those which keep anophelines at a distance ; removal of

TABLE G.

Year.	Almirante Division.				Costa Rica Division.			
	Tertian Cases.	E.A. Cases.	Percentage of E.A. Cases.	Hemoglobinuric Fever Cases.	Tertian Cases.	E.A. Cases.	Percentage of E.A. Cases.	Hemoglobinuric Fever Cases.
1931	6	92	93·88	3	3	466	99·36	28
1930	3	111	97·31	13	2	516	99·61	25
1929	17	211	92·54	6	61	702	92·00	21
1928	21	412	95·15	7	634	499	44·04	22
1927	111	231	67·54	4	653	484	42·57	39
1926	454	62	12·02	4	602	679	53·01	32
1925	808	40	4·72	9	262	367	58·35	17
1924	1,045	34	3·25	9	288	271	48·48	18
1923	981	86	8·06	11	739	173	18·97	15
1922	828	38	4·39	5	811	117	12·61	15
1921	467	15	3·11	9	550	97	14·99	12
1920	324	28	7·95	7	1,068	17	1·57	9
1919	448	43	8·76	7	703	19	2·35	11
1918	193	18	8·53	4	132	12	7·79	11
1917	149	33	18·13	7	177	82	31·66	2
1916	161	28	14·81	6	85	230	73·02	3
1915	135	42	23·73	8	48	255	84·16	7
1914	305	442	59·17	10	65	711	91·62	37

water which serves or might serve as breeding ground or its treatment with oil or Paris green; field surveys with treatment of infected persons, or the whole camp when infections exceed 25 per cent., with quinine and plasmochin. Measures such as screened quarters, destruction of adult mosquitoes, mosquito nets, mosquito repellants, are impracticable on the plantations. *A. G. B.*

i. RAINGEARD. La main-d'oeuvre au Kwango (Congo Belge). [**Labour in the Kwango District (Belgian Congo).**]—*Rev. Méd. et Hyg. Trop.* 1932. Jan.–Feb. Vol. 24. No. 1. pp. 21–48.

ii. REVUE DE MÉDECINE ET D'HYGIÈNE TROPICALES. 1932. May–June. Vol. 24. No. 3. pp. 133–148. With 1 map.—La main-d'oeuvre au Kwango (Congo Belge).—Lettre et rapports de la Cie du Kasai.

iii. HARMEGNIES. Rectification à propos de l'article du Dr. Raingeard.—*Rev. Méd. et Hyg. Trop.* 1932. July–Aug. Vol. 24. No. 4. pp. 180–182.

i. In a long article Dr. Raingeard indicts the management and treatment of native labourers in the Kwango district of Belgian Congo, where palm oil and rubber are produced in large quantities and where the author spent nearly four years as medical officer. He suggests that this district has been industrialized too rapidly and that the natives have suffered severely in the process. The regulations of the State made for their protection are disregarded. He speaks of their state as industrial slavery. He gives many details for which interested persons must consult his paper.

ii. The Compagnie du Kasai at Brussels writes that Dr. RAINGEARD was in its service and his charges concern the company, in which the State is not interested. They do not meet the charges directly but publish the report of the second semester of 1931 on trypanosomiasis and the report of the medical service for 1931 both by the Chief Medical Officer, Dr. HARMEGNIES.

iii. Dr. Harmegnies challenges Dr. RAINGEARD'S figures of trypanosome infection. He and a colleague have re-examined the population of 28,000 and find that it is increasing and that the births exceed the deaths. *A. G. B.*

CHESTERMAN (Clement C.). **The Training and Employment of African Natives as Medical Assistants.**—*Proc. Roy. Soc. Med.* 1932. May. Vol. 25. No. 7. pp. 1067–1074. (Sect. Trop. Dis. & Parasit. pp. 27–34).

This is an admirable article which no one concerned with its important subject should miss. The author writes from eleven years' experience at a mission station, Yakusu, Belgian Congo. He notes that the Colonial Governments in Tropical Africa are now addressing themselves seriously to the task of providing medical and sanitary services for natives in rural areas, so that the time is ripe for examining the situation as a whole as regards aims, policy and practice. The density of population in Tropical Africa is remarkably low. It rarely exceeds 15 to the square mile ; in Nigeria, Sierra Leone and Gambia it is about 50, whereas in Java it is no less than 686. The paucity of the population in spite of the pacification of the country is due to disease and the absence of hygiene. " The application of our knowledge is lagging too far behind the acquisition of it."

The difficulties are however great and are not merely geographical and administrative. There is the animistic belief in the spiritual causation of disease, either the witch or the shades of ancestors, which creates suspicion of measures which ignore these beliefs. The animist believes that he goes deeper than we to the cause of the trouble, for he isolates those who cause disease (witches, etc.) while we concern ourselves more with those who have it. The only solution is the training and employment of subordinate native staffs. Dr. Chesterman then deals with the objection that the negro's mentality is such that he cannot be trained or, when trained, trusted and suggests that the arrest of development at the age of puberty is a protective mechanism against an education illsuited to the actual needs of adult life. Simple medical education and practice are eminently related to his needs and environment and here he can make considerable progress while constantly kept in contact with the facts. What is needed is " the boy who has had the irreducible minimum of theoretical training, coupled with a sound practical experience in routine treatment and prevention, and who will thereafter work under regular supervision."

Passing over the section of the paper in which is described present-day practice in Sierra Leone, Gold Coast, Nigeria, the Sudan, Uganda, Tanganyika Territory, Nyasaland, we come to Belgian Congo. Here there are four official medical schools at the provincial capitals, in which about 150 students are undergoing a five year course to qualify as *infirmiers*. Proficiency in French and arithmetic is a prerequisite, three years are spent in theoretical and practical study, and two as a stage in some medical or sanitary formation before the final examination is taken. Chesterman feels that this limited training of native boys in rural hospitals whether government or private, as opposed to concentration at the large hospitals, is a sound policy.

He then describes his own experience at Yakusu. Students aged from 13 to 20 are admitted by competitive examination, the important subjects being French and arithmetic, they are provided with uniform

and rations and live in a hostel. The curriculum, which covers three years, is described. Practice and theory are combined from the outset and all boys are delegated in monthly rotation to the performance of duties suited to their year whether in the wards or out-patient department. The text book used is the "African Dispensary Handbook" [reviewed in this *Bulletin*, Vol. 27, p. 85]. After qualification boys are employed as monitors for a year at the training hospital before being sent to rural dispensary or medical post. They contract to give at least three years service and start at about £2 a month. In the rural dispensaries standard fees are charged, to cover the cost of drugs and dressings. The aim is to place these dispensaries at central market villages not less than 15 miles apart and to develop round them welfare centres visited once a week by a second assistant or trained woman. He finds that boys are capable of rendering faithful and efficient service even when visited only once or twice a year, but a three-month interval is preferable.

Dr. Chesterman goes on to make suggestions to those responsible for framing policies. He thinks that an A.D.M.S. should be appointed in each Colony with the supervision of medical education as his special duty. A new category of medical officers might be formed in the larger colonies looking to this work as a career. They should however be also practising at the rural hospitals. The A.D.M.S. (Education) would be responsible under the D.M.S. for the posting and advancement of the subordinate staff and for unifying the educational program, text books and examinations. Laboratory attendants, sanitary inspectors and dispensers would be his care. Advanced medical education the author does not discuss but he notes that in French West Africa, Belgian Congo and Tanganyika Territory higher education has either been modified after trial or not seriously contemplated.

Dr. J. Howard Cook who took part in the discussion said—

"With reference to policy in the training of the African, four principles should be recognised :—

"(1) Early training, pre-medical and welfare, and simple village hygiene, should be in the vernacular. A higher medical and nursing training in English. (2) Training should be intensive rather than extensive, aiming in the first instance at producing a few thoroughly reliable workers rather than a number of less reliable diplomates, and as the credit of the work will depend on the character of the men and women trained, every care should be taken in their selection and moral supervision and training. (3) All medical training should keep the right balance between theory and practice. The African who is trained in theory only, gets a swelled head and is often useless when it comes to practical work. (4) All elementary education should be correlated to the needs of rural village life." *A. G. B.*

Kassirskiy (J. A.). Die Methode der Knochenmarkpunktion bei tropischen Erkrankungen Mittelasiens. [**Examination of Bone Marrow in Tropical Disease.**]—*Arch. f. Schiffs- u. Trop.-Hyg.* 1932. Sept. Vol. 36. No. 9. pp. 492–495.

For obtaining a sample of bone marrow the author uses a Bier's needle with a guard 1 cm. from the point and he punctures the sternum at the level of the third intercostal space after preliminary local anaesthesia with 1·5–2 cc. of 1 per cent. novocaine. 0·5 cc. of marrow suffices for haematological and parasitological study.

The findings in various tropical diseases met with in Middle Asia (Russian Turkestan) are described :—In *malaria*, contrary to expectations, the marrow shows no peculiarities other than those visible in the

peripheral blood. In acute cases there is a marked myeloid reaction which, as the case becomes protracted and chronic, gradually disappears. The blood picture from the aspect of erythropoiesis is that of a mixed "normo-megalo-proerythroblastic reaction with preponderance of normoblasts."

In *kala azar* and *infant kala azar* sternum puncture is, according to the author, preferable to spleen puncture on the ground that the latter is a "blind" procedure which may be, though is not often, fraught with danger. Even if only a small amount of marrow is obtained the number of leishmania bodies seen is usually large. For children he uses a finer needle and, the bone being softer, no anaesthetic is required, since the operation is practically painless. One or two drops are sufficient for examination.

In *relapsing fever* no advantage is gained by examining the marrow; the findings run parallel with those of the peripheral blood. When the spirochaetes are no longer found in the blood they are absent also from the marrow and the blood change is, in general, that of a myeloid reaction both during the paroxysms and the apyretic periods. In *undulant fever* and septic conditions the author has obtained good growths with 5–10 cc. of marrow in 100 cc. of glucose broth.

In *sprue* the marrow is red and presents a megaloblastic picture in varying degrees. Even in anaemia of moderate severity a definite new formation of megaloblasts is obvious—megaloblasts of a lymphoid type and gigantoblasts with nucleus of a delicate structure, nucleoli and basophilic protoplasm, the basophilia varying in degree, and showing transitions from the initial forms (erythrogonia) to fully developed megaloblasts possessing ambliochromatic nuclei with the chromatin disposed in the "wheel" form and a polychromatophilic or orthochromatic protoplasm. It is interesting to note that this megaloblastic condition can be seen quite early in the disease when the anaemia is but slight, indicative of a specific reaction on the part of the bone marrow; nevertheless this does not necessarily persist but changes to a normoblastic reaction as recovery ensues.

In *pellagra* there is also a megaloblastic and proerythroblastic type of reaction, but not to the same degree as in sprue or in Addison's anaemia. The anaemia is of the hypochrome variety (60–70 per cent.) with absence of any haemolytic signs. This is of importance in distinguishing pellagra from conditions otherwise resembling it.

H. H. S.

RADSMA (W.) & STREEF (G. M.). **On Metabolism during Rest and Protein-Consumption in Europeans Living in the Tropics.**—*Arch. Néerlandaises de Physiol. de l'Homme et des Animaux.* 1932. Vol. 17. No. 1. pp. 97–123. [23 refs.]

An earlier study by one of these authors [Vol. 29, p. 235] showed that the basal metabolism of young Europeans resident in Batavia was lower than the normal figures given by BENEDICT and HARRIS and by DU BOIS. The study has been extended to include the measurement of urinary nitrogen excretion and to investigate the influence of the protein content of the diet on the basal metabolism. In the majority of subjects there is a lowering of the basal metabolism which in a group of 24 amounted to an average of 6·3 per cent. below the standard figures of Benedict. A particular group of nine subjects was investigated after a residence of 3 months and again after 2 years in Batavia and

although the metabolism figures remained approximately the same the difference for the standard values of Benedict had lessened from —10·6 per cent. to —7·5 per cent. owing to the fact that nearly all the subjects had lost weight in the 2 years. On the basis of the nitrogen excretion figures it is concluded that there are no serious differences in the protein consumption of the subjects of these tests and the American students who served for fixing the standard figures of Benedict and Harris. The lower values for the basal metabolism of Europeans in Batavia are not to be explained by any lessened protein intake. *M. E. Delafield.*

Radsma (W.), Streef (G. M.) & Klerks (J. V.). Over het zuurbase evenwicht bij tropenbewoners. Eerste mededeeling: Over de reactie der urine, in 't bijzonder der onder "basale" voorwaarden verkregen morgenurine, bij tropenbewoners. [**On the Acid Base Equilibrium in the Tropics. I. Reaction of the Urine, especially the Morning Urine under "Basal" Conditions in the Tropics.**]—*Geneesk. Tijdschr. v. Nederl.-Indië.* 1932. June 7. Vol. 72. No. 12. pp. 798–807. With 5 text figs.

The 24 hours' urine of Europeans in the tropics is more alkaline than that of the natives; the morning urine of Europeans is often alkaline (pH>7), this is rarely so in natives. Whether the difference in reaction is to be ascribed only to the more acid food of the natives (rice) or also to differences in the intermediate metabolism, respiration or perspiration is still to be decided.

The greater oscillations in the reaction of the urine in Europeans gives rise to interesting speculation as to their causes which, however, cannot be concisely reported upon. *W. J. Bais.*

Moustardier (G.). L'ulcère phagédénique des pays chauds et son traitement par l'émétique. [**Treatment of Phagedaenic Ulcer by Tartar Emetic.**]—*Bull. Soc. Path. Exot.* 1932. Apr. 13. Vol. 25. No. 4. pp. 333–339.

——. Traitement de l'ulcère phagédénique tropical par les injections intraveineuses d'émétique.—*Bull. Soc. Méd.-Chirurg. Indochine.* 1932. Apr. Vol. 10. No. 3. pp. 264–267.

The author, who writes from the convict station of Poulo-Condore in Indochina, says that tropical ulcer, almost always on the legs and feet, forms 10 per cent. of all diseases treated. The proportion is apparently raised by intentional ill treatment of slight wounds by the prisoners. It was found that treatment by 914 or sulpharsenol hastened recovery but these drugs were too expensive for general use. The author therefore tried another spirillicide, tartar emetic, in one per cent. solution, intravenously. On the day preceding the patient is purged. On the 1st day he receives 2 cc. of the emetic, on the 3rd 3, on the 5th 5 and on the 7th 10 cc., then every other day 10 cc., to a total of 1·2 gm. Concurrently the wound is cleaned with alcohol and covered with dressings kept moist with hydrogen peroxide. After the 2nd or 3rd injection the phagedaenic process is arrested and after the 4th or 5th has disappeared. As a rule 0·4–0·5 gm. of the drug is enough. One patient only had untoward symptoms and these were checked by the use of adrenalin. A brief account follows of 30 cases thus treated in which recovery ensued in from 7 to a maximum of 25 days. *A. G. B.*

SAYERS (Edward G.). **The Treatment of Tropical Ulcer by the Method of Dickson Wright.**—*Trans. Roy. Soc. Trop. Med. & Hyg.* 1932. June 30. Vol. 26. No. 1. pp. 49–54.

Dickson WRIGHT described his method of treating varicose and indolent ulcers of the leg in papers in the *British Medical Journal* and elsewhere in 1930 and 1931. The treatment seems to depend on two main propositions—that healing fails because of the grave circulatory difficulties in varicose ulcers, and that hermetical sealing with sticking plaster will promote healing better than anything else will. The method is described. The author, who writes from the Methodist Mission Hospital, Bilua, British Solomon Islands, used ordinary adhesive plaster and describes a series of ten cases in all of which success was attained. The leg is firmly encased from foot to knee in a 3-inch spiral bandage and ordinary activities are carried on. Discharge is disregarded. The bandage is changed once a week. Ulcers of all types were treated. The estimated cost is about a shilling a week. The method is particularly suited to natives, who tire of in-patient treatment or daily attendance. *A. G. B.*

ANDERSON (T. Farnworth) & ROBERTS (M. A. W.). **The Treatment of Ulcers by Skin Grafting.**—*East African Med. Jl.* formerly *Kenya & East African Med. Jl.* 1932. June. Vol. 9. No. 3. pp. 79–83.

Treatment by excision and grafting was described by BRENNAN [this *Bulletin*, Vol. 28, p. 839] and this method has been followed with the results noted—

No. of ulcers treated by skin grafting	154
No. of ulcers excised before grafting	112
Average total days in hospital of ulcer cases skin grafted ...	31·0
Average days in hospital after skin grafting (all cases) ...	19·6
Average days after skin grafting of cases in which whole graft took	14·9
No. of cases in which whole graft took	103
No. of cases in which more than half took	25
No. of cases in which less than half took	19
No. of cases in which graft failed	7

The average time spent in hospital by 100 cases treated by ordinary methods was 66·7 days ; the corresponding figure in the skin grafted series was 31·0. The ulcer and its base are removed with a sharp scalpel. For details the paper must be consulted. In one case the ulcer, 8 by 6 inches, was of 5 years' duration, and the patient had previously spent a year in hospital. Excision and grafting was followed by sound healing in six weeks. *A. G. B.*

ENZER (John). **The Treatment of Ulcers.** [Correspondence.]—*East African Med. Jl.* formerly *Kenya & East African Med. Jl.* 1932. Aug. Vol. 9. No. 5. pp. 148–149.

In reference to ANDERSON & ROBERTS' paper the author writes that of 160 cases treated by him by skin grafting in Mombasa 118 were successes, i.e. the whole graft took, and 12 days only were spent in hospital after the grafting. Protection with or without the simplest

dressing was all that was necessary. Where the Thiersch method fails he now employs "pinch grafting," small circular patches of whole skin distributed over the area. Here there were no failures.

A. G. B.

i. Loewenthal (Leonard J. A.). **Calcium Treatment in Tropical Ulcer.**—*East African Med. Jl.* formerly *Kenya & East African Med. Jl.* 1932. Aug. Vol. 9. No. 5. pp. 136–142.

ii. ——. **Tropical Ulcer as a Deficiency Disease: Treatment with Injections of Calcium.**—*Lancet.* 1932. Oct. 22. pp. 889–892.

i. In September 1931 treatment of tropical ulcer by injections of calcium was started at the Mbale hospital, and with such success that 4 weeks later the S.M.O. made it a routine measure first at the hospital and then at 7 sub-dispensaries. Grains 15 of calcium chloride in 10 cc. of distilled water are injected into the veins daily till cure is complete and, if a case proves resistant, the dose is doubled. Offensive odour is lost in the first week, sloughs in the second, and in the third there is a healthy granulating surface with rapidly extending epithelialization. Of the first 69 cases in only 11·6 per cent. was no improvement noted and this because the majority had disappeared in a week or less. The advantages of the treatment are cheapness, a month's supply of the drug for one patient costing less than 10 cents; convenience in that the whole treatment can be carried out by a native dresser; and employment of mass-therapy because outlying districts can be looked after by natives. Probably scraping and skin grafting would have shortened the treatment. The author goes on to discuss native diets showing that the bulk of his patients lived on plantains and plantain flour, which are deficient in both calcium and fat. He notes that tropical ulcer is never seen in chiefs, houseboys or prisoners, and in these classes the diet is more adequate. He also cites the Masai with a meat, blood and milk diet, and 3 per cent. ulcers contrasted with the Kikuyu, vegetarians, with 33 per cent., but notes that here as in the other cases absence of suitable protein may play a part. It is the calcium balance which is of such great importance. He refers to the work of Forbes Brown who obtained excellent results in ulcers by administration of parathyroid [see this *Bulletin*, Vol. 28, p. 839] and suggests that its action may be due to withdrawal of calcium from the bones and teeth. He realizes the importance of helminth infections and of food absorption from the alimentary tract, both of which subjects in relation to ulcer require study.

ii. The same author, whose experience is derived from a district in Uganda, says that though hospital records do not show a high death rate in this condition death, according to native chiefs, is the almost invariable result when the patients return to their villages. He thinks that in its mortality and morbidity tropical ulcer may take its place with malaria, plague and trypanosomiasis. McCulloch who studied the disease in Nigeria concluded—"Tropical ulcer is a dietetic ulcer, and the question of causative organisms need not be considered at all"; in this the author agrees. That it occurs sometimes in epidemics should not prejudice opinion in favour of its bacterial origin. It is found among the poorest classes almost exclusively: in his neighbourhood the Baganda are well-to-do and supplement their vegetable diet with meat and milk; natives of Ruanda come in to do the menial

work and live on a purely vegetable diet ; the vast majority of ulcers are seen in the Ruanda labourers. Clinically there is no appearance of inflammation ; the process is one of tissue necrosis on a large scale.

Five hundred cases have now received the intravenous calcium treatment which the author states is superior to all others both therapeutically and economically. *A. G. B.*

Fischl (Viktor). Chemotherapeutische Prüfung seltener Metalle. [**Chemotherapeutic Trial of Rare Metals.**]—*Ztschr. f. Hyg. u. Infektionskr.* 1932. Aug. 22. Vol. 114. No. 2. pp. 284–288. [12 refs.]

The results of prophylactic and curative trial of 18 trade compounds of 10 rare elements in experimental infection of mice with nagana (Prowazek) and European relapsing fever are here described and summed in a table. The substances shown to be active were—in relapsing fever ruthenium chloride, rhodium sodium chloride and two salts of osmium ; in trypanosomiasis " ruthenrot " and rhodium sodium chloride. Fourteen elements are now known to be spirochaeticidal (Vanadium, Gallium, Arsenic, Ruthenium, Rhodium, Silver, Indium, Antimony, Tellurium, Osmium, Platinum, Gold, Mercury and Bismuth) and eight trypanocidal (Gallium, Arsenic, Ruthenium, Rhodium, Indium, Antimony, Gold and Bismuth). He suggests that systematic trial of compounds of Palladium and Iridium would bring these into one or other list. *A. G. B.*

Kuhn (Philalethes) & Dombrowsky (Karl Heinz). Chemotherapeutische Untersuchungen mit Chinin-Weil bei der Vogelmalaria und der Pneumokokkeninfektion der weissen Mäuse. [**Chemotherapeutic Experiments with Quinine-Weil in Bird Malaria and Pneumococcus Infection of White Mice.**]—*Arch. f. Hyg. u. Bakt.* 1932. July. Vol. 108. No. 4. pp. 188–197.

Quinine-Weil is a compound of quinine base and acidum phenylcinchoninicum or atophan, a white powder readily soluble in water or better in a slightly acid medium, which quickly resolves into its two components. It has an advantage over hydrochloride of quinine in that it is quite tasteless. The authors conclude from their experiments that *Proteosoma praecox* infection in canaries can be cured by Quinine-Weil and by the hydrochloride, and that these drugs have no prophylactic but have a preventive action ; that is, given before they do not prevent infection but given from the day of inoculation they do. The treatment produced no untoward symptoms.

It is suggested that Quinine-Weil be employed in human malaria and in pneumonia. *A. G. B.*

Archives de l'Institut Pasteur de Tunis. 1932. July. Vol. 21. No. 1. pp. 154–173.—Fonctionnement des services de l'Institut Pasteur pendant l'année 1931. [**Activities of the Pasteur Institute of Tunis in 1931.**]

After an account of changes in personnel 8½ pages are devoted to the antirabic service. 1,686 persons presented themselves for treatment, a slightly lower figure than that of 1929 and 1930, but a figure which testifies to the constant non-observation of regulations by both the population of Tunis and the administrative agents. The

number of persons treated was 1,152, 7 of whom succumbed to rabies. Since its foundation the Institute has treated 20,583 persons, with 77 deaths, a fatality of 0·37 per cent.: this excludes deaths in the course of treatment or within 12 days thereafter. An ordinary method of treatment and an intensive method are described. Various data follow. The biting animals included 1,516 dogs, 87 cats, 25 solipeds, 21 ruminants and smaller numbers of other animals including 9 men. There were no paralytic symptoms in the course of treatment. The places of origin of the persons treated is given at length.

During the year 33,610 analyses were made, classed as microbiological, chemical, biological and parasitological. A study of this list gives a good idea of the diseases met with in Tunis. Other tables deal with the vaccines and serums produced for both human and veterinary use.

A. G. B.

Archives des Instituts Pasteur d'Indochine. 1932. Apr. No. 15. pp. 153–182.—Fonctionnement des Services Pratiques des Instituts Pasteur en 1931. [**Activities of the Pasteur Institutes of Indochina in 1931.**]

Saigon.—The number of examinations made in the Laboratory of Human Microbiology was 32,460, about three-fourths for the diagnosis and prevention of infective diseases. In the antirabic service 1,338 persons underwent a full course of treatment with one death, a mortality of 0·08 per cent. The diagnosis of rabies was confirmed by inoculation to rabbits in 85 instances out of 131. Nearly 3 million doses of antivariola vaccine were distributed, the product of 141 buffaloes. Of other vaccines 5,135,849 doses were prepared, including more than 4½ million for cholera. Of therapeutic serums 113,577 bottles were despatched for the treatment of tetanus, diphtheria, dysentery, plague, etc. and 8,000 doses of anatoxin for protection against diphtheria and tetanus, and tuberculin for diagnosis and treatment (Boquet and Nègre). In the antituberculosis service 4,573 examinations were made. Of BCG vaccine 101,287 doses were delivered, making a total since 1924 of 479,683 doses, sufficient for more than 159,800 children. The antimalarial service has a personnel of two doctors, an entomologist and an assistant, all Europeans, besides Indochinese. Its activities are described. Plasmodia were found in 2,668 cases (834 *P. vivax*, 331 *P. malariae*, 1,503 *P. falciparum*). This laboratory does some veterinary work, but the bulk of this is undertaken at Nhatrang.

The service of chemistry examines biological products, looks after water supplies, investigates food and drink for adulteration and conducts biological research. Thus it turned out in this year 200,000 0·25 gm. tablets of chaulmoogric acid for the treatment of leprosy.

Hanoi.—The activities of this Institute are similar to those of Saigon. Antivariolar vaccine, to the extent of 4,723,810 units were supplied to Tonking, North Annam, the Kwang Chau Wan territory, and to consular posts and missions in China. Here also antimalarial work is actively pursued: two doctors and an entomologist are employed.

The third Pasteur Institute, Nhatrang, is concerned entirely with animal pathology, chiefly rinderpest. *A. G. B.*

Riforma Medica. 1932. July 16. Vol. 48. No. 29. pp. 1115–1116.—L'idiosincrasia chininica e la sua cura. [**Idiosyncrasy to Quinine and its Treatment.**]

The manifestations of idiosyncrasy to quinine dealt with in this note are the scarlatiniform rash and urticaria with or without dyspnoea (fenomeni pseudo-asmatici). The pseudo-scarlatinal form is differentiated from true scarlatina by the absence of the other symptoms of the latter—high fever, vomiting, sore throat. Also, the rash appears suddenly and is generalized, it varies in intensity from time to time, and is accompanied by lowering of blood pressure and decrease in leucocytes.

Quinine urticaria, the author states, is easily diagnosed from ordinary urticaria; the "predominating signs are gastro-intestinal or respiratory disturbances" [but these may accompany urticaria of any origin]. Whichever form the rash assumes, if due to quinine it usually appears within an hour or so of the drug being taken, the dose has usually been large, and a repetition of the quinine will cause a fresh appearance of the rash. The author finds on enquiry that the symptoms arise when a dose of quinine is given after an interval which has allowed the preceding dose to be completely eliminated.

Hence, in treatment, to avoid such signs of idiosyncrasy, stress is laid on the administration of doses after the first within this interval, that is, before the preceding dose has been eliminated. If this cannot be done, he recommends that the reaction be provoked on the day on which there would not be a malarial paroxysm, and that the dose should be small. His own method of procedure is to give an injection of 10 cgm. of quinine; if after an hour there is no reaction he injects another 25 cgm., this will lead almost certainly to a reaction. If this is brief, the injection is repeated the same evening; otherwise it is postponed till the next morning and thereafter the injections are continued daily till the malaria is cured.

A useful note of warning is given. The symptoms described, though following the administration of quinine, must not be too hastily attributed to the drug; they may be due to the malaria itself and if treatment be withheld on the former assumption there may be a rude awakening in the supervention of malaria in a pernicious form. Under these circumstances (that the rash is due to the malaria and not the quinine) the patient will usually state that he has previously been able to take quinine without any such unusual effect.

H. H. S.

Lindén (Oskar). Weichteilverkalkungen nach Chinininjektionen. [**Calcareous Infiltration of the Soft Parts after Quinine Injections.**] —*Acta Radiologica.* 1932. Vol. 13. No. 1. pp. 57–63. With 5 figs. (4 on 2 plates). [28 refs.] English summary (5 lines).

These infiltrations as shown by X-rays are figured. They were seen in two cases in the pelvic region and upper thigh. Both patients had suffered from malaria about 15 years earlier and had received intramuscular and possibly subcutaneous injections of quinine, in one instance about 15 injections, in the other about 100 in the course of 5 years, resulting in abscesses and fistulae. In one the dose was known to be 0·5 gm. of the hydrochloride. *A. G. B.*

CHOPRA (R. N.) & MUKHERJEE (B.). **Indian Ipecacuanha.**—*Indian Med. Gaz.* 1932. Feb. Vol. 67. No. 2. pp. 88–90.

Another of the I.R.F.A. Indigenous Drug Series. Ipecacuanha is obtained from *Psychotria ipecacuanha* Stokes, a small plant indigenous to Brazil, the moniliform roots of which constitute the drug of commerce. Columbian ipecac is obtained from another species. Many Indian plants which have been regarded as substitutes are described or mentioned but none of these contains emetine. The ipecac plant was introduced in 1866 to experimental nurseries in India but it was not till 1921 that the report of the Bengal cinchona plantations sounded an optimistic note about its culture. Now 226,000 plants are grown in Mungpoo and other plantations with great success in Burma.

The Brussels Conference agreed that powdered ipecac should have an alkaloidal strength of at least 2 per cent., and this is the case with the Columbian and Indian roots as well as the Brazilian ; 70 per cent. in the Indian root is emetine against 72 in the Brazilian ; and the emetine content, it is reported, is now improved at Mungpoo. A quantity of Indian emetine was tested at Calcutta and was found to possess practically the same physical and chemical characteristics as a standard sample obtained from a reliable firm in Germany. Tried in cases of amoebic dysentery it gave very satisfactory results. The authors note that the Federated Malay States is now one of the chief sources of supply, which should encourage the Indian growers. The important factor in cultivation is moderately high altitude and little fluctuation in daily temperature.

[When the authors say that dysentery is one of the important causes of morbidity in the British and Indian Army they apparently fail to appreciate the fact that most of this is bacillary dysentery, in the treatment of which emetine is worse than useless. (See this *Bulletin*, Vol. 28, pp. 16–17.)] *A. G. B.*

EPSTEIN (David). **The Actions of Emetine and Cephaeline on the Circulation, Uterus and Intestine.**—*Quarterly Jl. Pharm. & Pharmacol.* 1932. Jan.–Mar. Vol. 5. No. 1. pp. 21–32. With 11 text figs. [12 refs.]

This work was done in the Department of Pharmacology, University of Cape Town ; the experimental animals were cats.

" It has been shown that emetine and cephaeline, when given in *large* doses, have a depressant effect on the heart, emetine being about one and a half times as active as cephaeline. The action is a direct one on the heart muscle and conducting tissue. The heart-beats are slowed and weakened until they gradually fade away, but various irregularities, *e.g.*, ventricular fibrillation and auriculo-ventricular heart-block may precede the final failure of the heart. While the effects described above are unlikely to be seen with therapeutic doses of the drugs, the sudden entry of strong concentrations of the alkaloids into the heart, as would occur if strong solutions were rapidly injected intravenously, might produce undesirable symptoms. It would appear safer, therefore, to avoid the use of the intravenous route when giving these drugs.

" Subcutaneous minimum lethal doses prove fatal after several days, death being largely due to respiratory failure. In these subacute or chronic cases of poisoning cephaeline is one and a half times as toxic as emetine. This type of poisoning is more likely to be seen clinically than the acute type."

" Both drugs produce marked dilatation of the coronary vessels.

" Both alkaloids in strong concentrations cause relaxation and depression of the uterus and intestine. Weak concentrations may cause contraction of the uterus." *A. G. B.*

CHOPRA (R. N.), DIKSHIT (B. B.) & CHOWHAN (J. S.). **Pharmacological Action of Berberine.**—*Indian Jl. Med. Res.* 1932. Apr. Vol. 19. No. 4. pp. 1193–1203. With 2 graphs. [11 refs.]

——. **Berberine and Berberine-containing Plants in Pharmacology and Therapeutics.**—*Indian Med. Gaz.* 1932. Apr. Vol. 67. No. 4. pp. 194–197.

Berberine is of interest to us because of its well-known action in oriental sore. It occurs chiefly in *Berberis aristata* and other members of the Berberis order and also in *Hydrastis canadensis* (golden seal) to the extent of nearly 2·5 per cent. A crude extract made from berberine plants is used in indigenous medicine and is sold in India under the name " rasaut." JOLLY used " rasaut " for oriental sore in 1911 but without giving an opinion on its value. One of the authors and Das GUPTA found that a dilution of 1 in 80,000 berberine inhibits the growth of *Leishmania tropica* in a modified NNN medium ; this action is specific. [See this *Bulletin*, Vol. 26, p. 752.] The authors tested berberine in malaria, giving the sulphate in doses of 3 to 5 grains three times a day. In nine instances no effect was produced, though quinine controlled the temperature and banished the parasites. In oriental sore a 2 per cent. solution injected at the base of the sore produces healing after 2 or 3 injections.

Here we have an account of the toxicity of the drug, its absorbability, its fate in the body, its effect on the cardio-vascular system and on the vasomotor centre, as well as on the respiratory system, uterus and bladder. *A. G. B.*

CHOPRA (R. N.) & MUKHERJEE (B.). **Thymol, Menthol and Camphor from Indian Sources.**—*Indian Med. Gaz.* 1932. July. Vol. 67. No. 7. pp. 361–365.

There is a great demand for these three drugs in India and large quantities are imported. The authors here examine the possibility of their manufacture in India on a commercial scale. The natural sources are described and the possible sources in India, but in each case there is a synthetic product which threatens the existence of the natural one and is likely to displace it in the near future. As a source of thymol they suggest the cultivation of eucalyptus from which piperitone, readily converted into thymol, can be produced at small cost.

A. G. B.

HILLENBRAND (Karl). Schwerhörigkeit nach Chenopodiumölvergiftung. [**Loss of Hearing after Oil of Chenopodium Poisoning.**] —*Muench. Med. Woch.* 1932. July 15. Vol. 79. No. 29. pp. 1152–1153.

A healthy woman operative aged 56 took at a physician's direction for " worms " 6 capsules of 16 drops each of chenopodium oil within 1–2 hours ; 12 worms were passed. Three weeks later she received

80 drops within a short period. An hour later she became giddy, vomited and had a rushing in the ears. These and other symptoms continued. Five weeks later there was still tinnitus and difficulty of hearing, as well as disturbance of equilibrium. She came to the throat and ear clinic at Tübingen where the lesion was found to be one of the internal ear. The author points out that with chenopodium the dose of 16 drops should not be exceeded, nor a total quantity of 38 drops (1 cc.) ; it must be followed by a purgative ; and must not be given on more than two consecutive days [see also this *Bulletin*, Vol. 23, p. 481 ; Vol. 25, p. 418 ; Vol. 27, p. 66.] *A. G. B.*

McKenzie (Alan). **Fatalities following the Administration of Intravenous Tartar Emetic.**—*Trans. Roy. Soc. Trop. Med. & Hyg.* 1932. Mar. 31. Vol. 25. No. 5. pp. 407–410.

In this paper four deaths are reported in African natives suffering from urinary schistosomiasis undergoing treatment with intravenous tartar emetic. In seven other cases toxic symptoms appeared followed by recovery. In all, except one patient who recovered, the antimony tartrate was placed in a sterile glass-stoppered bottle with sufficient chloroform to make 2·5 per cent. of the final solution ; this was periodically agitated for 48 hours, when sterile 0·5 per cent. sodium citrate in water was added to produce a 5 per cent. solution of tartar emetic. Each batch was used during a period of 14 days and rejected if the odour of chloroform had disappeared or if the solution was not clear. The four fatal cases were all treated with different batches of this solution.

The mild toxic features included tiredness, slow pulse, increased respiration, vomiting and generalized pains. Severe cases showed great mental restlessness, respiratory distress, and later coma, with loss of all reflexes. Three presented hepatomegaly and coffee ground vomiting, but only one was jaundiced. Most of the cases showed a leucopenia with a relative lymphocytosis ; band forms and metamyelocytes were numerous. At autopsy the liver was enlarged and pale, the spleen dark and congested, while the mucosa of the intestinal tract showed purpuric tendency in some cases. Microscopic section revealed fatty degeneration of the liver cells with destruction of cytoplasm, but little or no change in the nucleus. The kidneys presented congestion, toxic changes in the tubule cells and fine areas of haemorrhage. One batch of solution used in 6 of the 11 cases was investigated bacteriologically five weeks after it had been made up and non-motile bacilli were then found resembling somewhat Klebs-Loeffler bacilli. Two experiments made with this on rabbits showed that boiled and filtered solution proved more toxic than the unboiled solution, a finding which suggested that the toxic body may have been formed by dissociation of the antimony salt rather than by bacterial agency.

N. Hamilton Fairley.

De (M. N.). **Some Facts about the Incidence of Splenomegaly in Bengal.**—*Indian Jl. Med. Res.* 1932. Apr. Vol. 19. No. 4. pp. 1029–1033.

During a period of ten years, 1920–29, the total number of autopsies in the medical college group of hospitals, Calcutta, was 977. In 525 of these the spleens weighed from 200 to 1,500 grams, 100–150 gm. being taken as the weight of a normal spleen.

Number of cases.	Weight in grams.
356	200 to 500
118	500 to 1,000
39	1,000 to 1,500
12	above 1,500

In 203 the organs were felt to be fairly firm and the trabeculae were numerous and very prominent. In all there was generalized thickening of the capsule and in 198 definite evidence of perisplenitis with adhesions to the neighbouring viscera. These capular changes and adhesions were found very infrequent in true kala azar and these changes were independent of the size of the spleen.

Of the 525 cases 98 were diagnosed during life as malaria or kala azar but in at least 24 the autopsy and laboratory examinations failed to support the diagnosis. A series of 54 cases in which the weights varied from 600 to 2,700 grams was studied. Most came from certain districts of Bengal. They run a very chronic course with enormous spleen and liver all the time, and continue to follow their usual profession. They suffer from an irregular type of fever, labelled malaria, leishmaniasis, enteric, etc. Therapeutic measures are futile. A secondary type of anaemia always develops with marked diminution of leucocytes. They tend to develop fibrosis of the liver with ascites. They die of some intercurrent malady. At autopsy capsular changes and adhesions are very frequent.

" The histological examination of these spleens reveals a picture which is consistently found in every one of these cases, with minor modifications. In all of them, the splenic pattern tends to be lost and the malpighian follicles atrophied or totally destroyed. The entire pulp looks cellular, congested and full of many large phagocytic mononuclear cells. In some of the cases, the latter show intense red cell phagocytosis indicating an abnormally increased capacity of the organ for blood destruction. The capsules are thickened and the trabeculae very prominent containing much fibrous tissue. There is also a considerable increase of reticular fibres which appear fairly coarse and numerous when stained with Foot Bielschowsky's method."

During the period under review there were 36 cases of cirrhosis of the liver ; in 21 the average weight of the spleen was about 300 gm. and in 15 900 gm. ; the cirrhosis therefore may be classified in two distinct groups, with and without marked splenomegaly, and the author believes that these groups have separate aetiologies.

[One cannot tell from the paper whether the groups or series discussed are distinct or overlap, and, in the cirrhosis series, whether or not intermediate weights connected the 300 and 900 gram groups.]

A. G. B.

HEWER (T. F.). **Splenomegaly and Infantilism.**—*Trans. Roy. Soc. Trop. Med. & Hyg.* 1932. Aug. 11. Vol. 26. No. 2. pp. 139–146. [15 refs.]

" Seven cases of delayed sexual development are reported [from the Anglo-Egyptian Sudan] ; common factors were splenomegaly, exposure to malaria and bilharziasis, jaundice or a positive van den Bergh reaction, anaemia with low colour index, leucopenia, and eosinophilia. One of the cases attained maturity at the age of twenty-five without any regression of the other symptoms. There was no

apparent difference between these cases with infantilism and many others from the same neighbourhood with similar splenomegaly but normal development. The relation between the spleen, lipoid metabolism, the thyroid gland and sexual development is briefly discussed but no satisfactory conclusion achieved. These cases are reported largely to draw attention to a condition which may possibly be commoner than one would imagine, since the absence of any constant feature other than delayed development makes recognition difficult."

A. G. B.

Russell (Paul F.) & West (Augustus P.). **Charcoal as a Diluent for Paris Green in the Destruction of Anopheles Larvae. Larvicide Studies.**—*Philippine Jl. Sci.* 1932. July. Vol. 48. No. 3. pp. 291–297. With 3 figs. on 1 plate.

These authors find that a dust spray of wood charcoal with 1 per cent. Paris green kills over 80 per cent. of larvae in 4 hours, and all the larvae in 24 hours. The particles of Paris green must be small enough to be ingested by the larvae, must be distributed to all parts of the breeding area, must float long enough for the larvae to devour them, and must not be dangerous to other living creatures. In the Philippines charcoal is readily obtained, easily prepared (by pounding in a primitive pestle and mortar), floats longer than Paris green, is easily distributed and not expensive. The cost is about 0·003 dollar per pound. A litre of the mixture will treat 100 square metres of surface or along 100 metres of bank. *A. G. B.*

Ross (G. A. Park). **Bird-Lime for killing Mosquitoes.** [Correspondence.]—*Jl. Trop. Med. & Hyg.* 1932. Aug. 15. Vol. 35. No. 16. p. 256.

Dr. G. A. Park Ross, Assistant Health Officer, Union of South Africa, calls attention to the value of bird-lime as a means of killing mosquitoes and larvae in tanks, wells and pools. Grass or small twigs are smeared with it and thrown on the surface of the water. It is said to be as efficacious as paraffin. For pools, sticks with short branches, which project just clear of the water, are used.

A. G. B.

Ginsburg (J. M.). **Airplane Oiling to control Mosquitoes.**—*Science.* 1932. May 20. Vol. 75. No. 1951. p. 542. [Summarized in *Rev. Applied Entom.* 1932. Aug. Vol. 20. Ser. B. Pt. 8. p. 176.]

"Since in the swamps and marshes of New Jersey, where the larvae of the prevailing mosquitos are primarily bottom feeders, control by arsenical or other dust is either ineffective or not practicable, experiments were carried out during the summer of 1931 to determine the possibility of applying oil or other liquid larvicides by means of an aeroplane. Two 50 U.S. gal. tanks were installed in the forward cockpit and connected to a steel pipe, 3 inches in diameter, extending along the bottom of the fuselage and terminating just below the rudder in a cross pipe, 7 feet long and $1\frac{1}{2}$ inches in diameter, perforated with holes, $\frac{1}{8}$–$\frac{1}{4}$ inch in diameter, from which the liquid flows. A valve, placed at the rear end of the longitudinal pipe and operated by the pilot, controls the flow of oil. The aeroplane flies as low as possible, and the liquid flows out as a fine spray, depositing a thin

film over the breeding areas. By this method both pyrethrum larvicide and oil were tested. The results showed that on a still day a killing film is deposited. In two areas, practically complete kill of larvae and pupae was obtained. About 8 acres of a meadow were covered with a killing film of oil in about 40 minutes, the amount required for this being about 140 U.S. gals., and the cost of the application less than that of hand oiling."

PONS (R.). Essais de désinsectisation durable du sol sablonneux des cases au Sénégal. [**Disinfection of the Sandy Floor of the Huts in Senegal.**]—*Bull. Soc. Path. Exot.* 1932. June 8. Vol. 25. No. 6. pp. 539–541.

Fleas are extraordinarily numerous in the sandy floors of these huts; 150–300 specimens may be taken in one in a few hours. The genus is in nearly all cases Synosternus, probably an important plague carrier for specimens collected in a plague-stricken hut infected 6 guineapigs out of 12 injected, and in a village built on laterite in which plague had never occurred no house fleas could be found. After consultation with the local branch of the Standard Oil Company the author selected for trial fuel oil with two-thirds of gas oil. The penetration of this mixture is such that a thickness of 1 cm. impregnates to a depth of 15 cm. The author thinks 2 cm. should be applied. The price per sq. metre is 8 francs, giving 50–60 francs per hut. The mixture is applied with a watering can with a rose and adsorption is so complete that the product does not stick to the shoes or feet; it is however advisable to spread a little dry sand on the surface. Four huts have been thus treated and after visits 8, 21, and 30 days later no fleas could be found. *A. G. B.*

DILIBERTO (Ugo). Nota sulla colecistopatia da lamblia. [**Lamblia in the Gall-Bladder.**]—*Riv. Sanitaria Siciliana.* 1932. Jan. 15. Vol. 20. No. 2. pp. 113–114, 117-119. English summary (8 lines).

The patient, a man 18 years of age, had suffered at intervals during 8 years from attacks of abdominal pain, particularly in the epigastrium, more frequent during the last 8 months. There was neither vomiting nor fever. Blood examination showed red cells 3,800,000, white 4,900 per cmm., Hb. 73 per cent., C.I. 0·9. The only other physical signs were those obtained by the duodenal sound. Two of three samples of bile contained Giardia. One presented no macroscopic changes, but microscopically vegetative forms of the parasite were seen in the deposit; the other was darker, with flocculi which revealed massed Giardia and a few leucocytes.

Serge's treatment with naphthalene, 50 cgm. daily in 3 cachets, was given for a week. The symptoms cleared up entirely and repeated duodenal sounding since has given negative findings. There has been no return of the symptoms though several months have passed.

Absence of fever and leucocytosis exclude actual cholecystitis, but the author is of opinion that Giardia can give rise to a "cholecystopathy," analogous to an "appendicopathy" associated with Enterobius without definite appendicitis. *H. H. S.*

KOUWENAAR (W.). **Gastric and Duodenal Ulcers in Sumatra's East Coast and their Pathogenesis.**—*Far Eastern Assoc. Trop. Med. Trans. Eighth Congress, Bangkok, 1930. Dec.* pp. 587–595.

It is generally held that gastric and duodenal ulcers are very rare in the tropics. The author's material is derived from 3,161 autopsies

made in the Pathological Laboratory and hospitals in Deli, Sumatra ; nearly all were indentured labourers.

TABLE I.

	Number of post mortem examinations.	Number of ulcers and scars.
Chinese men	1,370	151=11%
Javanese men	1,269	8=0·63%
Javanese women	399	4=1%
Tamils	123	6=4·9%

A second table gives the number of ulcer patients in age-decennia : this brings out that the frequency of ulcer increases with age, but that the increased liability of the Chinese is present at all ages. The author notes that gastric ulcer in European men on the East Coast of Sumatra is common. He suggests that this greatly increased frequency of gastric and duodenal ulcers in Chinese men as compared with Javanese men is of importance for the study of its causation and the possible reasons for the difference are discussed. Nothing of significance results and the problem remains to be solved. In the discussion SNIJDERS remarked that the specialists on tropical disease in Holland find high figures for gastric and duodenal ulcers among patients long resident in the Dutch Indies. *A. G. B.*

DUBOIS (A.). Note sur la tension artérielle chez les indigènes congolais. [**Arterial Tension of Congolese Natives.**]—*Ann. Soc. Belge de Méd. Trop.* 1932. June 30. Vol. 12. No. 2. pp. 133–136.

The author has tested the arterial tension of 200 apparently healthy natives of the Mabudu tribe in the region of Pawa, a Red Cross Station in Belgian Congo. He used the auscultation apparatus of Korotkov, with which normal subjects in Europe give maxima of 13–14 cm. ; the author's own tension in Africa was 13·5.

The results were as follows :—

4 between 9 and 10·5 cm. (tension very feeble)
30 ,, 11 and 12·5 cm. (tension low)
95 ,, 13 and 14·5 cm. (tension normal).
49 ,, 15 and 16·5 cm. (hypertension light).
20 ,, 17 and 20 cm. (hypertension clear).
2 upwards of 20 cm. (20·5 and 25) (hypertension very strong).

Of 50 natives of age apparently below 40, 7 had a tension reaching or exceeding 15 cm. (14 per cent.) ; of 150 natives over 40, 64 reached or exceeded 15 cm. (42·6 per cent.). He concludes that of Congolese natives living a primitive life a fair number have arterial tension above the normal, but usually moderate. These persons are, as in Europe, relatively old. The clinical manifestations of this state are inconspicuous. The only evident cause is age. [But see DENNISON, this *Bulletin*, Vol. 27, p. 162, where bloodpressure in African natives (Kenya) *diminishes* with age.] *A. G. B.*

RUSSELL (H.) & TAYLOR (C. J. S. O.). **A Case of Sickle Cell Anaemia.**—*West African Med. Jl.* 1932. Apr. Vol. 5. No. 4. pp. 68–69. With 2 coloured plates.

Sickle cell anaemia has been chiefly described in negroes of the United States. ARCHIBALD has reported a case in an Arab boy in the Anglo-Egyptian Sudan [this *Bulletin*, Vol. 24, p. 66].

A case seen in Accra is here described. An African boy of 14 of Ga parentage was admitted for debility. He complained of feeling tired and of breathlessness. He had marked anaemia, thumping heart sounds and a systolic murmur at the base of the heart and in the mitral area. The liver was palpable two fingers breadth below the costal margin; spleen not enlarged. A thin blood film showed spindle and oat shaped red cells, and in a cover glass preparation ringed and kept for two hours most of the cells were crescentic.

Blood count.—Red cells, 1,940,000. White cells, 13,000. Haemoglobin, 50 per cent. Colour index, 1·3. Lymphocytes, 49·5 per cent. Polymorph leucocytes, 37 per cent. Large mononuclear cells, 6 per cent. Eosinophil polymorphs, 6 per cent. Normoblasts, 3. Basophil polymorph, 1–1·5 per cent. Myelocytes, 2. Platelets 1 to 3–4 red corpuscles.

The Wassermann was negative.

The parents and 4 other children of this family were examined. In the cover glass preparations there was sickling in the father's blood and that of a girl of 3.

Sickling has been noted three times in natives of the Gold Coast post mortem, and is probably not rare. *A. G. B.*

ARGAUD (R.). Ostéolyse ainhumique. [**A Case of Ainhum.**]—*Bull. Soc. Path. Exot.* 1932. Apr. 13. Vol. 25. No. 4. pp. 323–325.
DELANOË (E.). Un cas d'aïnhum.—*Ibid.* pp. 322–323. With 1 text fig.

Each of these describes a typical case of ainhum which would hardly call for special notice except for the fact that the first patient was a negress and the condition is comparatively rare in women and that the origin of the lesion dated from a definite injury, cutting on a stone, followed by suppuration. The second, in a male of 21 years, is designated cryptogenic on the grounds that it was discovered accidentally after death from pneumonia and no history was therefore obtainable.

The morbid histology—thickening of the dermal layers with osteoporosis and absorption of bone—was that usually present. *H. H. S.*

VAN DEN BRANDEN (F.) & APPELMANS (M.). La pathogénie de l'ainhum. [**The Pathogeny of Ainhum.**]—*Ann. Soc. Belge de Méd. Trop.* 1932. June 30. Vol. 12. No. 2. pp. 161–172. [25 refs.]

The authors describe two cases seen at Leopoldville and discuss the etiology in some detail without reaching any conclusion. The pathogenic mechanism of ainhum, they say, is still obscure. Its frequent bilateral localization suggests the intervention of a local factor such as the repeated traumatism to which the bare-footed are exposed, and of a general predisposing factor which eludes them. A page of references is sketchily presented. *A. G. B.*

HUARD. Les myosites et les pseudo-myosites en milieu colonial. [**Myositis and Pseudo-Myositis in the Tropics.**]—*Ann. de Méd. et de Pharm. Colon.* 1932. Jan.–Feb.–Mar. Vol. 30. No. 1. pp. 5–33. [4 pages of refs.]

In explanation of his title the author points out that true myositis, verified microscopically, is rare and what is often termed myositis, namely abscess developed more or less deeply in the juxta-muscular connective tissue, should be called pseudo-myositis. He reviews his subject at length under the headings—(a) Myositis and pseudo-myositis without clinical septicaemia ; (b) the same with septicaemia ; (c) how to examine a case of myositis, and (d) treatment.

Under (a) he considers that the relation between filaria and multiple abscesses remains to be proved and that an amoebic origin, demonstrated in a well-reported case by SHARP and MORRISON [this *Bulletin*, Vol. 22, p. 347] is very rare. The septicaemic states considered under (b) are those due to staphylococci, streptococci, pasteurella, pneumococci and typhoid, the first of which is by far the most important ; he regrets that no animal experiments such as those of ROSENOW have been made in the tropics. In section (c) the author describes at length both clinical and laboratory methods of examination ; this should be consulted by those who have to diagnose these conditions. The whole paper, though some of it is elementary, is worth perusal. The numerous references are nearly all French. *A. G. B.*

MONTEL (J.). Un nouveau traitement de la fièvre ondulante par des dérivés de la mélitine et de l'abortine : les formomélitines et les formabortines. [**A New Treatment of Undulant Fever by Formolized Melitin.**]—*Marseille-Méd.* 1932. Apr. 25. Vol. 69. No. 12. pp. 537–543.

Melitin or abortin is prepared by growing the corresponding strain of Brucella in broth for 3 weeks, and filtering through a Chamberland candle. To the filtrate 0·3 per cent. of formol is added, and the mixture is placed in the incubator for 2 months. The product, described as formomelitin or formabortin, is stated to be non-toxic to patients suffering from undulant fever and to possess definite immunizing properties that render it valuable for therapeutic purposes. Treatment is carried out by inoculation of quantities varying from 1–5 cc. ; even larger doses may be used. There is practically no local reaction, and the temperature does not rise more than 1°C. Case histories are given of patients treated by this method after other means, including vaccines, had failed, and apparently very favourable results have been obtained. *G. S. Wilson.*

SNIJDERS (E. P.), KOUWENAAR (W.), MAASLAND (J. H.) & WOLFF (J. W.). Onderzoek over rhinoscleroom op Sumatra (2de Mededeeling). [**Rhinoscleroma in Sumatra.**]—*Nederl. Tijdschr. v. Geneesk.* 1932. July 30. Vol. 76. No. 31. pp. 3733–3740. With 2 maps & 1 plate. English summary.

This condition of rhinoscleroma has all the characters of a chronic inflammatory affection in which pure cultures of a capsulated bacillus *Klebsiella rhinoscleromatis* can be obtained from the deeper tissues. An investigation into the locality from which came a series of 11 sporadic cases between 1918 and March 1931 showed that 10 were Bataks.

On the strength of this finding an expedition was planned and made to the island of Samosir in the Toba lake. Here and in the neighbourhood 22 new cases were found. All the facts furnished by these new cases—family relationship, closeness of contact, etc.—pointed to the disease being a slowly infective one. A parallelism is drawn between rhinoscleroma and leprosy as regards duration of contact for infection, the presence of a characteristic bacillus and endemicity. Rhinoscleroma, so far as facts are available, seems to be a disease of an old and primitive people, of which the Bataks are a remnant and possibly also the inhabitants of the Chota Nagpur plateau in British India. Thus the problem becomes to some extent the interesting one of geographical pathology and anthropology. It might be further investigated from this point of view in other localities.

W. F. Harvey.

HERMITTE (L. C. D.). **A Practical Contribution to the Bacteriology and Treatment of Otitis.**—*Trans. Roy. Soc. Trop. Med. & Hyg.* 1932. Aug. 11. Vol. 26. No. 2. pp. 189–194.

The author, who has practised for five years in the Seychelles, gives his experience of the part played by *Pseudomonas pyocyanea* in lesions of the external and middle ears frequently met with there. In the period 1927–31 he examined 233 patients for ear trouble and the present paper deals with 56 cases of diffuse inflammation of the external ear, especially those refractory to all forms of local treatment. A short account is given of 21 cases. All were of long duration, in all there was pyocyaneus infection and autopyocyaneus vaccine was almost invariably curative. The bacillus is grown on agar. For detail the paper must be consulted. He suggests that this condition is of wide occurrence in the tropics. *A. G. B.*

SHERIF (M. A. F.). *Erythria spicata* **and** *Glinus lotoides* **(D.) or** *Molugo glinus* **(A. Rich.) as Antidotes for Scorpion Stings.**—*Jl. Egyptian Med. Assoc.* 1932. July. Vol. 15. No. 7. pp. 460–464.

A popular remedy for scorpion stings in Egypt is what is known in the market as "El Teriak El-Masri," which is prepared from *Erythraea spicata* (Gentianaceae) and *Mollugo glinus* (*Glinus lotoides*) Aizoaceae. The drug was tested subcutaneously in rats and rabbits against scorpion venom. In rats it neither modified the symptoms nor saved life ; in rabbits it appeared as if life was slightly prolonged, but death was not averted. *A. G. B.*

CLUNIE (T.). **Appendicitis in Fijians.**—*Jl. Trop. Med. & Hyg.* 1932. Aug. 15. Vol. 35. No. 16. pp. 241–242.

The author cites a case of acute appendicitis in a Fijian, whose appendix was removed by Dr. HARPER and a case of his own in a Fijian child aged 8. Aubrey MONTAGUE in an experience of nearly 30 years in Fiji never saw a case of appendicitis in the native. During the last 20 years the Fijian diet on plantations and in centres like Suva has changed, meat, rice and white bread taking the place of native roots. It is suggested that the absence or diminution of cellulose and fibre favours the onset of appendicitis. [There seems however little evidence that appendicitis is anything but a rarity.] *A. G. B.*

Moise (Regolo). Studi sugli eritroconti (V. Schilling) nell'anemia perniciosa, nella malaria ed in altre malattie. [**Erythroconts in Disease.**]—*Ann. di Med. Nav. e Colon.* 1932. July–Aug. 38th Year. Vol. 2. No. 1–2. pp. 385–396. With 6 figs. on 3 plates. [15 refs.] English summary (8 lines).

The author observed in the blood of certain patients with severe anaemia of malarial and other origin, rod-shaped bodies, resembling Bartonella, but reacting differently to stains such as Nile Blue, Giemsa, and May-Grünwald, and differing also from azurophil nuclear residues (Jolly bodies) and the basophile granules of lead poisoning. These bodies have been named erythroconts by V. Schilling. With Giemsa stain they are coloured blue or blue-grey, whereas Bartonella are bright red. They are always intracorpuscular and rarely more than one per corpuscle. The author does not believe they are artefacts or due, as has been suggested, to folds in the corpuscular structure. He did not find them in several cases of leukaemia, myelocytic or lymphocytic, in the anaemia of cancer, in a case of lead poisoning and in patients with subtertian malaria, but they were present in 14 cases of pernicious anaemia (not in 4 others who were improving with treatment by liver) and in anaemic patients with benign tertian infection. *H. H. S.*

Müller (H.). Histoplasmose in Oost-Java. [**Histoplasmosis in East Java.**]—*Geneesk. Tijdschr. v. Nederl.-Indië.* 1932. July 5. Vol. 72. No. 14. pp. 889–895. With 4 figs. on 2 plates. [15 refs.] German summary.

The case described by the author is the first of its kind which has been diagnosed outside America. A 7-year-old Javanese boy from the interior had been ill for some time and suffered from continuous fever, abdominal cramps and vomiting. He was greatly emaciated, with enlarged hard glands in various situations but especially in the neck region. A hard movable tumour mass was palpable in the abdomen. The reactions of v. Pirquet, Wassermann and Sachs-Georgi were negative and there was no evidence of leukaemia. At death it was found that all the lymph glands, peripheral as well as internal were quite white, friable, enlarged but discrete, and could be shelled out. No caseation was evident. A handsbreadth above the ileocaecal valve was an ulcer with undermined edges. Microscopically the affected tissues showed spherical Gram-staining organisms, with an appearance as of a surrounding unstained capsule and no internal structure. They were 1 to 3 μ in size and were contained within the cytoplasm of large macrophages of undoubted reticulo-endothelial type. The diagnosis lay between kala azar and histoplasmosis. Kala azar does not occur in the Netherlands Indies nor was there any indication of the presence of the distinctive blepharoplast or kinetonucleus of *Leishmania donovani*. The positive coloration by Gram was another differential feature. In considering the nature of the organism the author refers to its affinity with the blastomycete *Cryptococcus farciminosus*, which is the causal organism of epizootic lymphangitis in horses. It could not however, be cultivated, whereas the cryptococcus is easily grown. Epizootic lymphangitis is common

in Java, but on the other hand, it does not exist in America where the first cases of histoplasmosis were discovered. This case forms only the sixth of the total number hitherto described.

W. F. Harvey.

Benjamins (C. E.). Over Hooikoorts (Pollenosis) in Indië. [**On Hayfever (Pollenosis) in the Netherlands Indies.**]—*Geneesk. Tijdschr. v. Nederl.-Indië.* 1932. Aug. 2. Vol. 72. No. 16. pp. 1016–1027. With 13 figs.

The author intends to determine whether the so-called "rhinitis spastica tropica" is not, at least in a number of cases, actually an allergic condition like the real European hayfever. The observation in Holland of 3 patients who suffered from spastic rhinitis in the Netherlands Indies showed that they were allergic against the pollen of various tropical Graminaceous species, including rice pollen (*Oryza sativa*), as well as against those of European grass species. Further study of this subject in the tropics especially in regard to Ambrosia and Artemisia species, is needed. Anti-allergic treatment by means of pollen extracts looks more promising than the symptomatic treatment practised so far.

With pollen of various palm trees positive allergic reactions were seen in a European hayfever patient. In this patient as well as in patients from the tropics the pollen of Oreodoxa (? cabbage palm tree) caused no reaction. Yet since long in the tropics some connexion has been recognized between the flowering of this palm and the rhinitis attacks, which fact the author ascribes to the mechanical irritation of the mucous membrane by the sharp edged Oreodoxa pollen, which is shown in illustrations. *W. J. Bais.*

Giglioli (George). **Paratyphoid C Case Reports from British Guiana.** —*Jl. Trop. Med. & Hyg.* 1932. Oct. 1. Vol. 35. No. 19. pp. 290–297. With 4 charts. [20 refs.] [Summary appears also in *Bulletin of Hygiene.*]

This article is largely clinical, several cases being described in detail with their respective temperature charts. The author has previously recorded cases and outbreaks in British Guiana among employees of the Demerara Bauxite Co. to which he was medical officer. He notes that infection by *Bact. paratyphosum C* is widespread and that in the tropics conditions prevail which give opportunity for, if they do not actually favour, explosive outbreaks, and it therefore calls for attention every whit as much as other enterica infections.

The ordinary T.A.B. vaccine is prophylactically powerless against it and the quadruple T.A.B.C. is necessary. This was first used in the Serbian army during the Great War. Dr. Giglioli made it and employed it extensively in Demerara; during the period 1928–30 more than 1,100 persons were inoculated and though cases continue to occur among the non-vaccinated population, none of the inoculated has developed the disease. *H. H. S.*

CASTELLANI (Aldo). **Epidemiological Notes on Some Tropical Mycoses.** —*Proc. Roy. Soc. Med.* 1932. Sept. Vol. 25. No. 11. pp. 1605–1614 (Sect. Epidemiol. & State Med. pp. 79–88).

The author deals with tonsillomycoses and bronchomycoses, Madura foot, tinea cruris, tinea flava, tinea nigra, pinta, tinea imbricata and a few other skin conditions. Most of the information is to be found in CASTELLANI and CHALMERS' "Manual of Tropical Medicine."

A. G. B.

DO AMARAL (Zepherino). Eléphantiasis scrotal. [**Scrotal Elephantiasis.**] —*Rev. Sud-Américaine de Méd. et de Chirurg.* 1932. Mar. Vol. 3. No. 3. pp. 223–227. With 2 text figs.

In this Brazilian case microfilariae were sought on three occasions without success. The scrotal swelling was regarded as "elephantiasis nostras"; it followed an injury to the perineum. *A. G. B.*

BARNSLEY (A.). Anaesthesia in the Tropics. Basal Narcosis with "Avertin."—*Malayan Med. Jl.* 1932. Mar. Vol. 7. No. 1. pp. 1–12. With 4 charts. [26 refs.]

BEIJNEN (G. J. W. Koolemans). Diarrheeën van tropischen oorsprong.—*Nederl. Tijdschr. v. Geneesk.* 1932. Sept. 3. Vol. 76. No. 36. pp. 4190–4199.

BERTI (Edoardo). Appunti di noso-geografia sulla Msellata (Tripolitania).—*Arch. Ital. Sci. Med. Colon.* 1932. May 1. Vol. 13. No. 5. pp. 296–313. With 5 text figs. English summary (5 lines).

CASTELLANI (Aldo). Brevi cenni storici sulla medicina tropicale dai tempi antichi ai nostri giorni.—*Policlinico.* Sez. Prat. 1932. July 18. Vol. 39. No. 29. pp. 1117–1130. With 15 figs.

DEL VAL (José). Cirugia en los tropicos.—*Medicina Países Cálidos.* Madrid. 1932. Sept. Vol. 5. No. 5. pp. 353–370. With 13 figs.

ELMES (B. G. T.). A Case of Abdominal Lymphosarcoma with Associated Splenomegaly.—*Trans. Roy. Soc. Trop. Med. & Hyg.* 1932. Aug. 11. Vol. 26. No. 2. pp. 195–197. With 3 figs. on 1 plate.

HUISMAN (R. G. J. P.). De sympathico-diaphtherese volgens Doppler (phenolisatie der arterien) en hare toepassing bij de behandeling van ulcera cruris (tropica).—*Geneesk. Tijdschr. v. Nederl.-Indië.* 1932. Oct. 11. Vol. 72. No. 21. pp. 1435-1439.

LACQUET (Albert M.). Experimental Pathology of the Liver. VIII. Effects of Carbon Tetrachloride on the Normal and on the Restored Liver after Partial Hepatectomy.—*Arch. Pathology.* 1932. Aug. Vol. 14. No. 2. pp. 164–176. With 6 figs. [18 refs.]

MEIKLEJOHN (A. P.), PASSMORE (R.) & PETERS (R. A.). The Independence of Vitamin B Deficiency and Inanition.—*Proc. Roy. Soc.* Ser. B. 1932. Sept. 1. Vol. 111. No. B 772. pp. 391–395. With 3 figs.

ROBIC (J.). Notes sur les infections à déterminations gastro-intestinales observées à Tananarive, pendant la saison chaude 1931–1932.—*Bull. Soc. Path. Exot.* 1932. July 6. Vol. 25. No. 7. pp. 790–795.

ROBIN (L. A.). Perfectionnement apporté â une méthode d'enrichissement des selles.—*Bull. Soc. Path. Exot.* 1932. July 6. Vol. 25. No. 7. pp. 700–702.

Seraglia (P.). Contributo allo studio sulle condizioni sanitarie dei Baluba del Lomami.—*Arch. Ital. Sci. Med. Colon.* 1932. May 1. Vol. 13. No. 5. pp. 283–295. With 11 text figs. English summary (6 lines).

Tessitore (Carlo). Note di nosografia generale della zona di Bumba (Congo Belga).—*Arch. Ital. Sci. Med. Colon.* 1932. June 1. Vol. 13. No. 6. pp. 355–366. English summary (3 lines).

Tsuchiya (H.). A Practical Staining Method for Intestinal Protozoa.—*Jl. Lab. & Clin. Med.* 1932. Aug. Vol. 17. No. 11. pp. 1163–1164.

Zottner. Coloration du sang et des hématozoaires par la méthode de Stévenel, modifiée.—8 pp. With 7 figs. on 4 plates. Travail du Laboratoire de Recherches du Service de l'Elevage du Maroc.

REVIEWS AND NOTICES.

LANE (Clayton) [M. D., Lond., Lt.-Colonel, I.M.S. (retd.), formerly in Charge of the Darjeeling Ankylostome Inquiry, and co-opted Member of the Sanitary Board, Bengal, and of its Hookworm Disease Committee]. **Hookworm Infection.**—pp. xiv+319. With 36 figs. 1932. London, etc.: Humphrey Milford, Oxford University Press. [25s.]

Colonel Clayton Lane's long association with hookworm research in relation to the health of the individual and the community entitles him to write a book on the subject from the point of view of a successful specialist.

While the various aspects of the subject are discussed very fully, direct diagnosis, treatment and prevention receive especially detailed consideration. The author accepts the following records of animal hosts other than man for *A. duodenale*, gorilla, tiger, pig and the civet; for *N. americanus* dog, cat, rhinoceros and pig. Rough geographical limits are given as 36°N. and 30°S. latitudes, with great extension beyond these limits in special cases such as mines, where conditions of an artificial kind are favourable to extracorporeal development. The conclusion that necator infections tend to predominate in the hotter, and ancylostome infections in cooler climates, is rejected on the finding that at 5,000 to 7,000 feet in Darjeeling there is a pure necator infection, while ancylostomes appear at lower, warmer, altitudes. Many fallacious conclusions such as the above are held to have resulted from the use in experiments of untrapped cultures.

With reference to extracorporeal life, the evidence discussed leads to the conclusion that while eggs only survive about three months, larvae in favourable conditions will live long enough to carry on infection from year to year, and may be vigorous enough to do so; the author does not agree with those who hold that larvae in soil die out in six or seven weeks, and who base their anti-hookworm campaigns on this assumption. Soil which appears quite dry has been proved to harbour larvae, and in any case the author points out that it is the condition of the places used for defaecation which is of paramount importance, and that these are frequently shaded, and moist, especially where water is used after defaecation.

The author strongly advocates the use of direct centrifugal floatation, and D.C.F.F. which is "Direct centrifugal floatation pushed to finality." His opinion of it is frequently expressed in some such terms as the following. "The real value of certain concentrative diagnostic techniques as measured by egg counts is set out in Fig. 26, the delivery of D.C.F.F. being taken as 100." It will be concluded that he has complete confidence that for accuracy and reliability his method cannot be surpassed at the present time by any method in use, and the evidence which he brings forward to support these high claims certainly appears convincing.

Treatment receives very full consideration and the author's conclusions are not favourable to the more modern systems of hookworm therapeutics, either as regards their safety or success. Thymol, beta-naphthol, oil of chenopodium, carbon tetrachloride, tetrachlorethylene and hexylresorcinol and various mixtures of drugs are each discussed in a critical manner. The conclusion reached will come as a surprise to those who believe that hookworm therapy has advanced with each new drug introduced. "On all the exact evidence which has been obtained and marshalled, *particulated* thymol is the drug of election for the individual, or for the mass, treatment of hookworm infection"; those who are not acquainted with the method described by GILES so long ago as 1890 will find it discussed here.

As regards mass treatment of large populations, on the basis of positive findings which may apply to only a small percentage of the people, a system referred to by the author as "herd treatment," many readers will agree with his emphatic hostility to it. "I hold the system to be inexpedient, unscientific, and immoral." He rightly argues that in

the long run it pays to treat the individual who requires treatment as an individual, and as a patient whose life is just as valuable to him and his friends, as it would be if he did not happen to suffer from hookworm infection.

It is not to be expected that the author will instantly succeed in convincing all those who hold opinions different from his own. There should thus be a good, if somewhat controversial, future before this book, since it is difficult to see how any serious worker can afford to be without its guidance, or to ignore its helpfully critical spirit. *B. Blacklock.*

Wakil (A. W.) [M.B., Ch.B. (Cairo), Dip.Hyg. (Camb.), D.T.M. & H. (Camb.), etc.] **The Third Pandemic of Plague in Egypt. Historical, Statistical and Epidemiological Remarks on the First Thirty Two Years of its Prevalence.**—Egyptian University. Faculty of Medicine. Publication No. 3. 169 pp. With 27 charts. 1932. Cairo : Egyptian University.

The first chapter of this book gives a brief account of the history of epidemic plague with particular reference to Egypt. The experience of that country is, in some striking respects, similar to that of Europe. One has, in the contemporary records, the same tendency to wild exaggeration of the numbers killed, but also the same evidence of very long intermissions between pandemic periods. Thus after the epidemic of A.D. 719 no other occurred for nearly 300 years. The present epidemic period began after a free interval of more than 50 years. One factor of the dwindling of plague after 1844 is suggested to have been the change in the trade routes from the east (culminating in the opening of the Suez Canal in 1869) and the contemporaneous freedom of India from epidemic plague. The next chapter is devoted to the origin of the epidemic period which began in 1899—*i.e.*, after plague had again become widespread in India. The most plausible of the theories of origin seems to be that grain sacks—presumably containing infected rats or fleas—reached Alexandria by rail from Suez. In 1899 Alexandria alone was involved. The next chapter deals with the progressive dispersion of plague in the first 6 years of the epoch ; the number of deaths increased from 45 in the first to 501 in the last of the six years. Chapter 4 provides statistical data for the whole period 1899–1930. The peak years have been 1907 (914 deaths), 1911 (1,041 deaths), 1916 (828 deaths), and 1923 (726 deaths). In 1930 there were 109 deaths. The author is disposed to attribute the relative mildness of the present plague cycle to an improvement of environmental and sanitary conditions rather than any biological peculiarity of the rodents which form the reservoirs and distributors of infection. Abundant data of the local distribution of plague are provided. A point of interest is that the clinical pneumonic type of plague, which in Egypt as a whole forms about 10 per cent. of the total, is much more frequent in Upper Egypt than elsewhere. A plausible explanation is that, owing to differences in the irrigation system, many of the inhabitants of Southern Egypt are forced to remain idle during March-May (the plague season). Domestic overcrowding, due to this circumstance, together with the return home of villagers who had migrated to the infected ports of Alexandria, Port Said and Suez in search of work and came home to die may be responsible.

The author next takes up the study of the distribution of different kinds of rat and their flea infestation. The next chapter discusses the special features of plague in Cairo and Alexandria. Cairo has enjoyed a remarkable immunity ; in the period 1899–1930 only 16 cases of plague with 11 deaths have been recorded. One factor of this immunity seems to be biological. Since the middle of the last century weasels have bred in large numbers in the city and have no doubt helped to keep down the rat population. Alexandria on the other hand has suffered a good deal from plague. It seems probable that plague is kept alive in Alexandria amongst the local population of rats although bacteriological proof is not easy to

obtain. Although thousands of rats have been caught and dead rats collected, sometimes from houses in which plague in man has occurred, positive bacteriological findings have been rare.

Professor Wakil has collected in this volume a great deal of information and presented it clearly. It is a pity that his modesty did not allow him to round off the work with a general review of the evidence and conclusions deducible from it. *M. Greenwood.*

THAYSEN (Th. E. Hess) [M.D., Senior Physician, St. Elisabeth's Hospital, Copenhagen]. **Non-Tropical Sprue. A Study in Idiopathic Steatorrhoea.**—pp. iii + 258. With 39 figs. 1932. Copenhagen: Levin & Munksgaard. London: Humphrey Milford, Oxford University Press. [10s. paper; 12s. 6d. cloth.]

The author's avowed purpose is to describe the clinical picture of non-tropical sprue, to demonstrate that it is identical with sprue occurring in the tropics and to stimulate interest in the many problems still unsolved. With this end in view he presents a detailed study of the clinical features and the pathological anatomy of both and discusses the diagnosis, pathogenesis and treatment. A work such as this makes an exceptionally wide appeal. It is of interest to tropical practitioners whether abroad or at home, for it is all important for them to distinguish true sprue from sprue-like diseases, particularly with the bias which all those who have practised in the tropics unwittingly have to regard anyone with steatorrhoea who has lived abroad as a case of sprue. Again, it concerns the general practitioner at home, since any one of them may meet with men from the tropics who have suffered from sprue in the past and may relapse, or who develop sprue after their return from abroad; or thirdly, they may meet with other conditions presenting some at least of the symptoms of sprue, *e.g.*, pellagra (of which several cases have been recorded in England), pancreatic disease, coeliac disease, though to mention the last savours of a *petitio principii*, since the author maintains that Gee-Herter's disease (GEE 1888, HERTER 1908), tropical and non-tropical sprue, grouped together as "idiopathic steatorrhoea," are closely related and possibly identical, not a triple personality but one and the same condition which if it occurs in the tropics is regarded as sprue, outside the tropics as "non-tropical sprue" in adults and as Gee-Herter's disease in children.

The author mentions all the cases he has been able to find in the literature; those reported by HOLMES and STARR, and PORTER and RUCKER (in U.S.A.), by SCHEER (Holland), SCHÄFFER, WILFERT, HEGLER (Germany) ROSENDAHL (Norway), HOLST, GEILL, JÖRGENSEN and WARBURG and the author himself (Denmark), GLOOR, and MEYER (Switzerland) and MANSON-BAHR (England). A table of 34 is given, and in addition he discusses CHVOSTEK's and UMBER's cases diagnosed as "functional pancreatic achylia" and SCHÖNEMANN's patient with "pluriglandular insufficiency with tetany" and fatty diarrhoea due to pancreatic lesion.

Each of the symptoms is analysed in turn and its significance discussed. The similarities between tropical and non-tropical sprue (including Gee-Herter's syndrome) are the fatty diarrhoea, stomatitis, emaciation and anaemia; in both the fat absorption is lowered in the bad periods, protein absorption is normal, there is a normal respiratory quotient, often increase in basal metabolism, a low blood sugar curve after ingestion of glucose, and a disturbance of mineral metabolism. There are, of course, individual variations; nevertheless "it is not only the chief symptoms . . . that are the same, but a detailed analysis of these symptoms shows a most convincing congruity between non-tropical and tropical sprue," and again later the author writes, "In non-tropical sprue one does not meet with a single symptom that is not encountered in cases of sprue developed in the tropics and vice versa, and this correspondence of the symptomatology . . . becomes even more conspicuous as the individual symptoms are analysed more thoroughly."

To speak in a little more detail of a few special points: It would seem to the reviewer that rather too much stress is laid on a history of a previous acute or chronic intestinal disturbance. This can be of little value; it is probably equally common in those who do not develop sprue-like symptoms later; it certainly is so abroad. Again, too much is made of the age factor, especially if the thesis is sustained that the coeliac affection in children and adults and non-tropical and tropical sprue are one and the same.

Dr. Thaysen's view of the anaemia is very sound and judicial. He remarks on the tendency to a shifting back and forth between hypo- and hyper-chromatic types; hence the classification of these anaemias is somewhat arbitrary and if the patient be observed for a sufficient length of time either type may be found. The chemical findings of the blood—urea, uric acid, creatinin, non-protein nitrogen, cholesterol—were found by him to be the same in non-tropical as has been recorded by MACKIE, FAIRLEY, ASHFORD and SERRA in tropical sprue.

As regards the pancreas he quotes BEGG and CAMMIDGE to the effect that "chronic pancreatitis is a frequent complication of tropical sprue" but since autopsy usually reveals a normal gland or very slight changes "these are to be taken as secondary phenomena" both in non-tropical as well as tropical sprue. With this probably all investigators now agree, and analyses of the fats in the stools and the ratio between the neutral fats and fatty acids also bear this out.

The author's discussion on the endocrine glands in relation to the symptoms of sprue is good and very fair. He gives priority of place to the parathyroids and states "FALTA assumes that there is a certain interaction between the parathyroids and the thyroid." When discussing SCOTT's theory of parathyroid action in regulation of the serum calcium changes in tropical sprue he does not mention [perhaps he was not aware of it] a fact strongly in favour of this interaction, viz., that when he (SCOTT) was carrying out experiments on his own case he was able during the stage of recovery to reproduce the symptoms at will by stopping parathyroid and substituting for 3 days or so thyroid extract. He was able to verify it in another patient also. It was not part of this theory that a "decrease of serum calcium is characteristic of sprue" (p. 89), but that in cases of average severity, and in early or recovering cases, the total was about normal [this is stated in a footnote later, p. 177] and that the fault was a mal-regulation of it due to functional parathyroid inadequacy. In these 34 cases of non-tropical sprue 11 showed symptoms attributable to some endocrine hypo- or dys-function. Tetany was present in 9; twice complicated with goitre. [Perhaps the latter was cystic and therefore of deficient not excessive action.] That "calcium absorption is lowered in certain cases is evident from the presence of osteoporosis [illustrative of this is a beautiful plate] and from the low calcium content in the blood in cases without tetany," but "calcium elimination in the faeces is increased" states the author; may not both, however, be due to increased calcium abstraction?

In the section dealing with aetiology, three main theories are discussed: Infection, Avitaminosis and the Constitution Theory. Dr. Thaysen concludes that the first is the least tenable, though it may help to explain the rarity or absence of sprue in some tropical countries, *e.g.*, Africa, Jamaica, and its frequency in others, as India, Ceylon, China, Porto Rico. As regards the second, the evidence is at present conflicting. The third is adduced to explain the rarity of sprue in natives [but recent accounts show that it is probably not so rare as has been believed]; at all events the term is too vague to have a tangible meaning.

The presentment and argument of the theories of pathogenesis are put with all fairness; the question of steatorrhoea naturally is dealt with in greater detail, whether it arises from defective splitting of fats in the intestine, rapid evacuation, defective absorption or abnormal excretion

from the bowel. In this connexion the fat content of the blood is a matter calling for more thorough investigation.

Morbid anatomy and histology are largely negative ; there is the same lack of characteristic changes in the intestine in non-tropical as in tropical sprue. A series of microphotographs of the intestine is reproduced, but such, even under the best conditions, are very inadequate for showing the finer morphological changes.

There is little calling for comment in the chapter on diagnosis. In chronic pancreatogenous fatty diarrhoea the elimination of nitrogen is almost constantly increased, seldom in sprue ; chemical analysis of the faeces also will reveal the difference. Hyperchromatic anaemia together with fatty diarrhoea is strongly suggestive of sprue. Steatorrhoea occurs in tuberculosis of the mesenteric glands ; gastro-jejuno-colic fistula causes fatty diarrhoea, emaciation and anaemia [see this *Bulletin*, 1932, vol. 29, p. 463] ; the distinction from pernicious anaemia has repeatedly been pointed out. Pellagra, if skin lesions are inconspicuous and intestinal symptoms and glossitis are prominent, may constitute a difficulty, but fat absorption in pellagra is usually normal, the anaemia is not of the hyperchromatic type, and we usually have the history of seasonal relapse. There is a good table of the chief diagnostic points between pancreatogenous fatty diarrhoea, mesenteric tuberculous adenitis and non-tropical sprue.

The vexed question of treatment is summarized with judicial restraint ; with the exception of diet, it is largely symptomatic, as is but natural, where the cause is obscure. Calcium checks the diarrhoea and renders the stools less acid ; hydrochloric acid is serviceable if there is achylia ; for hypochromatic anaemia iron, for hyperchromatic, liver, vitamin B (marmite). The author is of opinion that the influence of diet has been over estimated because success has followed any of at least five modes : 1. Milk and no meat (Manson, Low) ; 2 Meat and no milk (Cantlie, Elders) ; 3. Pure carbohydrate (Collin) ; 4. High protein, low fat and carbohydrate (Fairley) ; 5. Pure fruit (v.d. Burg).

In conclusion, we have in this book a very difficult problem most ably argued. Under present circumstances the utmost that anyone can do is to marshal fairly the evidence for and against and let those with experience draw their own conclusions. Certain small points of distinction exist between cases of tropical and those of non-tropical sprue, but whether these are sufficiently marked to warrant separation into two entities is doubtful ; there is much greater difference between, say, Oroya fever and verruga peruviana. All those working at this much debated question will be grateful to the author, who throughout this book has given full credit and prominence to British investigators, a courtesy which is by no means general among continental writers. *H. H. S.*

Blacklock (D. B.) [M.D., Professor of Parasitology, Liverpool School of Tropical Medicine, University of Liverpool]. **An Empire Problem: the House and Village in the Tropics.**—100 pp. 1932. The University Press of Liverpool. London: Hodder & Stoughton Ltd. [3s. 6d.] [Review appears also in *Bulletin of Hygiene.*]

The prosperity of trade between Great Britain and the overseas dependencies of the Empire is closely connected with the health of the natives who inhabit those dependencies, and produce the raw materials needed in the home country. The progress of scientific research has thrown a flood of light upon those diseases which render life in the tropics precarious, and are responsible for such a heavy mortality and morbidity amongst the native races. These are in the main preventable diseases, and science has placed in our hands specific remedies for their cure or prevention. Professor Blacklock, within the compass of a hundred brief pages, gives a masterly survey of the principal diseases which make his tropical home so

dangerous to the native, and his environment so uncomfortable. The book is written for non-medical readers in simple language, and is primarily intended to catch the eye of the larger trading companies, who in their business relations come into close contact with the natives with whom they trade. However valuable the discoveries placed in our hands by medical and scientific research, they are useless unless they can be practically applied to the needs of the native inhabitant. This is the "empire problem" of which the book speaks. Its solution depends on the co-operation between Government and the agents of commerce.

Professor Blacklock, in an extremely interesting way, classifies the dangers which threaten life, health, and economic efficiency in the tropics under three main heads: (1) diseases connected with the houses, (2) those associated with the village water supplies, and (3) those arising out of the existing imperfect methods of disposal of human excreta. Under the first category the potential dangers of dirty and cracked floors in harbouring fleas, flies, ticks, and bugs are explained in terms of the diseases they bring; then the roof with its rats and mosquitoes, and the diseases introduced by them; and finally the ravages caused by termites in the wooden supports of the native hut. In the second category the water-borne diseases, cholera, typhoid, bacillary and amoebic dysentery, ankylostomiasis, bilharzia, and the guineaworm are reviewed, and the life-history of their causal organisms sufficiently described to show the line that preventive measures should take. Other diseases, indirectly dependent on uncontaminated water, are reviewed—such as malaria, elephantiasis, yellow fever, and sleeping sickness. Finally the importance of providing suitable latrines is emphasized, and the principles of hygienic and simple latrines of the bore hole and large pit types are explained. The whole message of the book is summed up in a clear recapitulation-chapter, dealing with practical measures by which commerce can co-operate with Government in vastly improving the health and trading capacity of the native.

The book would be improved by a few illustrations, but it would be hard to imagine any material improvement in the way the subject matter is treated. Some of the descriptions, such as the ravages of rats, and their ingenuity of adaptation, the destructive power of termites, the helplessness of the naked child to escape myriads of disease-producing bites from scores of enemies, and the misery of unlighted villages are so graphic as to be unforgettable.

The measures outlined in this book will go a long way towards solving the empire problem, but one ventures to think that it will never be completely solved along these lines. The total number of white men is so infinitesimal in comparison with the native population of Africa, that Africa can only really be reformed by Africans. Ultimately the solution must be found by training African doctors, sanitary inspectors, welfare workers, nurses and the like, and stationing them at strategic centres to teach the rural population. Lastly, every white immigrant in the country, Government agent, missionary, trader and settler alike, should have a course of elementary medical and hygienic training sufficient to enable him to be an apostle of hygiene.

J. Howard Cook.

ORIEL (G. H.) [M.D. (Cantab.)]. **Allergy.** With Foreword by H. W. BARBER, M.B., B.Ch. (Cantab.), F.R.C.P.—pp. ix + 78. Pocket-Monographs on Practical Medicine. 1932. London: Bale, Sons & Danielsson, Ltd. [2s. 6d.]

This book is reviewed in *Bulletin of Hygiene*, 1932, Vol. 7, p. 600.

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